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AMERICAN JOURNAL OF OPHTHALMOLOGY

VOLUME 31 DECEMBER, 1948 NUMBER 12

THE FASCIA OF THE ORBIT*

ITS ANATOMY AND CLINICAL SIGNIFICANCE

RICHARD G. SCOBEE, M.D.
Saint Louis, Missouri

Much of the anatomy of the normal fascia of the orbit has been well known for more than a century and yet the average ophthalmologist is still not entirely familiar with certain portions of it. Various subdivisions of this fascia play a prominent role in the success or failure of the surgical treatment of heterotropia. Some parts may contain developmental anomalies which probably play an important role in the etiology of heterotropia.[1] It would, therefore, seem worthwhile to review and summarize the known facts about the fascia of the orbit and at the same time emphasize the clinical significance of certain sections.

On any map of the nation's highways, each road has a different number, the equivalent of a name. The same road is also part of the system of highways of the nation and connects with other highways which also have numbers or names. Each road is an integral part of the highway system. In similar fashion, many subdivisions of the fascia of the orbit bear different names but nevertheless all comprise the fascia of the orbit and any one part connects with one or more other parts. Since the fascia of the orbit is more or less continuous, in a sense, the apex of the orbit is as good a place as any to begin the consideration.

*From the Department of Ophthalmology, Washington University School of Medicine, and the Oscar Johnson Institute. This study was made under a contract with the Office of Naval Research as Project N6onr-202, Task Order I, NR 141-022.

THE DURA MATER

The intracranial portion of the optic nerve is sheathed in the meninges. As the nerve passes through the optic canal at the apex of the orbit, the meninges are rather closely pressed around it. The orbital portion of the nerve is much more loosely ensheathed.

At the point where the dura mater emerges from the canal of the optic nerve at the apex of the orbit, it divides into two layers. The inner layer continues along the optic nerve to the junction of that structure with the eyeball. The outer layer spreads out to cover the bony walls of the orbit and become known as the *periorbita.*

The function of the periorbita is similar to that of periosteum elsewhere. At the anterior end of the orbit—the orbital margin—the periorbita is confluent with the periosteum covering the bones of the face; at the rim of the orbit where these two differently named but similar layers of fascia meet, they become thickened to form a ridge known as the *arcus marginale.*[2]

This ridge of fascia marks the exact line of the orbital margin and from it arises the *septum orbitale* of the eyelids. This latter structure is also a part of the fascia of the orbit.

THE ANNULUS OF ZINN

Returning to the apex of the orbit where the dura mater of the optic nerve divides into an outer layer—the periorbita—and an

inner layer—the dura of the orbital portion of the optic nerve—there is a roughly circular tendinous cord lying between the two layers immediately after they have become distinct and separate at the apex of the orbit. This tendinous cord is known as the *annulus of Zinn* and it is rather intimately fused with both the periorbita and the dura of the optic nerve. The annulus has a very interesting history.

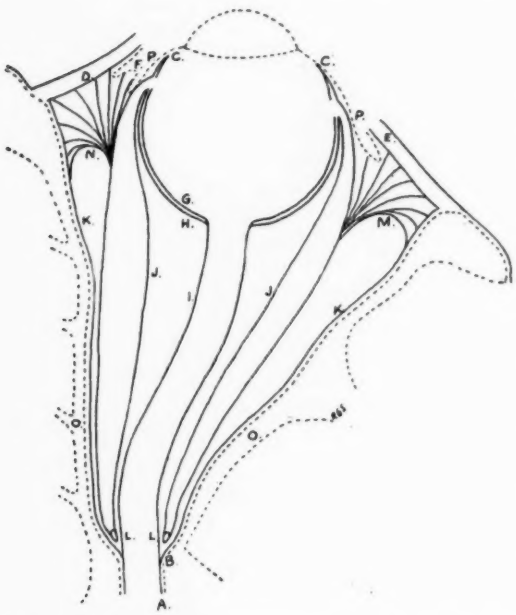

Fig. 1 (Scobee). Diagram showing only the fascia of the orbit (in solid lines). Bone, cornea, and conjunctiva are represented in dotted lines.

A. Meninges of optic nerve in optic canal.
B. Point of division of dura mater into periorbita and optic nerve sheath.
C. Point of fusion of Tenon's capsule with sclera.
D. Medial palpebral ligament.
E. Lateral palpebral ligament.
F. Lacrimal caruncle.
G. Visceral layer of Tenon's capsule.
H. Parietal layer of Tenon's capsule.
I. Dural sheath of optic nerve.
J. Muscle sheath.
K. Periorbita.
L. Annulus of Zinn.
M. Check ligament of lateral rectus muscle.
N. Check ligament of medial rectus muscle.
O. Bone of orbital wall.
P. Conjunctiva.

It is apparently not generally known that Zinn[3] described ". . . a ligament common to the inferior, internal, and external recti" only. He did not describe the entire circular structure as we know it today.

Sappey[4] described Zinn's structure as ". . . a fibrous cord which is attached to a very small fossa situated beneath and external to the optic foramen. This cord speedily divides into three slips—a middle, which goes to the inferior rectus; an inner, to the internal rectus; and an external, to the external rectus. Of these three bands the middle is the largest."

Lockwood[5] added to Sappey's remark that ". . . it may be added that the fasciculus, which belongs to the external rectus, is prolonged up the posterior border of the muscle, and ends by blending with the origin of the superior rectus."

It is obvious that the structure which Zinn described was in reality only the lower half of what is now referred to as the annulus of Zinn. The technique of dissection in the time of Zinn was well standardized in the case of the orbit and consisted in an approach to the orbital contents from above, the roof having been removed.

This external view of the muscle cone from above, the fat having been dissected away, revealed the fibrous appearing orbital surfaces of the superior rectus and the upper halves of the medial and lateral recti; from this approach, these fibrous outer surfaces of the muscles mentioned appeared to blend intimately with the dura mater of the optic nerve.

The technique then called for division of the superior rectus at its origin, followed by division of the optic nerve within its bony canal; both structures were next lifted upward and pushed aside. This maneuver exposed the inner surfaces of the medial and lateral rectus muscles and of the inferior rectus. From this aspect, the tendinous band across the inferior half of the optic foramen was exposed as the obvious point of origin of the three muscles listed.

It remained for Lockwood[5] to obtain a frozen orbit and divide it in a plane passing through the center of the pupil and the center of the optic foramen. When the inferior surface of this upper half of the orbit was inspected and the optic nerve pulled from its bed in the canal, the inner surfaces of the superior rectus as well as of the superior halves of the medial and lateral rectus muscles were then exposed to view and a tendon similar to the one Zinn had described below was easily visualized. This upper tendon has since been correctly called the *upper tendon of Lockwood* while the lower one is known as the *lower tendon of Zinn*, and the two together make up what is somewhat incorrectly but widely known as the *annulus of Zinn*.

The point of nomenclature here is one of purely academic interest and yet the story invites attention because it shows how often inaccuracies have crept into medical literature with the passage of time. A prime example of such an inaccuracy is the widely held notion that Helmholz invented the ophthalmoscope when it was in fact Babbage, an English mathematician, who first conceived the instrument.[6]

The capsule of Tenon

Consider now the dura which ensheathes the optic nerve in its orbital course. This membrane fuses with the fascial capsule which encases the globe—the *capsule of Tenon*.

Tenon[7] discovered the capsule which bears his name in 1803 and reported it in 1806. Surprisingly enough, or perhaps not so surprisingly after all, it was then forgotten for many years and Hyrtl[8] said that Malgaigne[9] finally brought it again to notice but called it by the name of the *orbital aponeurosis*.

In 1841, Bonnet[10] wrote an elaborate description of it and emphasized its surgical importance; his work was so well received that many began to speak of the structure as the capsule of Bonnet. In the same year, Ferrall[11] published a paper on the *tunica vaginalis oculi* in which he rediscovered the original capsule of Tenon; quite obviously, he was familiar with the work of neither Tenon nor Bonnet.

Since that time, many have described the capsule of Tenon, prompting Dwight[12] to observe somewhat cynically that ". . . the complications of this membrane are limited only by the perverted ingenuity of those who describe it."

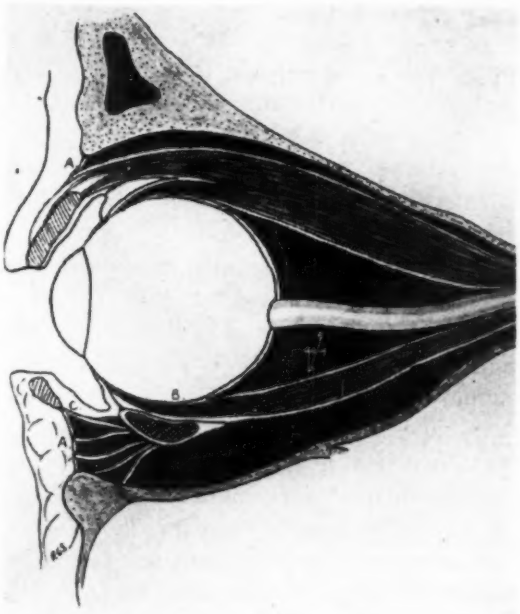

Fig. 2 (Scobee). Schematic vertical section through the orbit. The extensions of the suspensory ligament of Lockwood, particularly the one to the inferior tarsal plate (C), are shown, as is the suspensory ligament (B). The orbital septum is shown (A) superiorly and inferiorly. Note the close association of the levator and the superior rectus throughout most of their course.

The capsule of Tenon encases the globe from the limbus to the optic nerve, has a yellowish color and a closely matted appearance. The anterior third of the capsule is closely related to the bulbar conjunctiva under which it lies but from which it may be easily separated to within about 2 mm. from the limbus. In vertical sections, the anterior

portion of the capsule seems to blend with the superior and inferior conjunctival fornices. At these fornices, the capsule divides into two portions; one goes to the tarsal plate and the other runs to the sclera with the bulbar conjunctiva.

The middle third of the capsule is pierced by the six oculorotary muscles passing toward their scleral insertions. At the point where each penetrates the capsule, a prolongation of the capsule is sent backward along each muscle—*the muscle sheaths.*

The posterior third of the capsule is thinner than the anterior and middle thirds and is in contact with and loosely adherent to the orbital fat.

Whitnall[2] is of the opinion that connective tissue strands, hardened by alcohol or formalin and isolated by dissection from fat and neighboring structures, assume in many regions ". . . unwarrantable distinctions as membranes, ligaments, or capsules." He believes that if a firm body, such as the globe, revolves against a cushion of fatty tissue, the fat will be pressed away and the connective-tissue meshwork supporting it will be condensed into a membranoid surface. He states that this is what has happened in the case of the capsule of Tenon, agreeing that its posterior surface is connected with the meshwork of orbital fat, and that even in his most careful dissections, the posterior aspect of the capsule of Tenon is ragged and perforate. The earlier investigators, however, specifically stated that they used either fresh specimens or frozen ones rather than those which had been immersed in fixatives; hence Whitnall's objections to modern techniques would not seem applicable to the works of the older anatomists.

Schwalbe[13] described the capsule of Tenon as being divided into a visceral and a parietal layer. Of the two, the outer or parietal layer is the thicker and more easily defined and is the portion usually described as the capsule of Tenon. It is also referred to by some as the *fascia bulbi.*

The visceral layer, which covers the sclera and the region about the tendinous insertions of the oculorotary muscles, is thin and delicate. The space between the two layers is known as the *space of Tenon* or the *suprascleral lymph space of Schwalbe.* Delicate connective tissue fibers are found extending directly and obliquely across this space between the two layers.

In tenonitis, Lockwood[5] believed that it was the visceral layer of the capsule which was primarily involved, since the visceral layer is very similar to episcleral tissue.

There is universal agreement that the capsule of Tenon posteriorly is rather firmly adherent to the sclera around the point of exit of the optic nerve and that it fuses with the dural sheath of the nerve at their junction. Lockwood reported that the parietal layer of the capsule is composed of white fibrous tissue and elastic tissue while the visceral layer is mostly loose areolar tissue. Bonnet referred to the visceral layer as *subconjunctival fascia.*

Where the oculorotary muscles enter the capsule on the way to their scleral insertions, there are six visible openings. Each muscle is lightly attached to its own opening by the visceral layer of the capsule of Tenon. Lockwood described a band of fibers embedded in the capsule crossing the posterior edge of each opening and called these bands the *intracapsular ligaments* (fig. 3). There have been some interesting suppositions about these structures.

Ferrall[11] supposed that the intracapsular ligaments acted as pulleys over which the muscles worked, but this seems very doubtful. Whitnall[2] flatly stated that ". . . there are no pulley bars or intracapsular ligaments, and no need for them."

Lockwood,[5] however, described the intracapsular ligaments and said of those of the medial and lateral rectus muscles that their inferior ends were attached to the suspensory ligament (of Lockwood) and thus held firmly in place. He had a particularly interesting observation about the intracapsular ligament of the superior rectus, saying that

". . . it is quite lengthy, its inner extremity being fastened to the trochlea while the outer is attached to the wall of the orbit just above the lateral rectus muscle. Its oblique direction alters decidedly the pull of the tendon of the superior rectus which must turn nasally after passing through its opening in the capsule to reach its insertion in the sclera."

In speaking of the inferior oblique, Lockwood wrote that ". . . its aperture is just behind and lateral to that for the inferior rectus in the capsule, but it is separated from it by the intracapsular ligament which the suspensory ligament forms for that muscle and, in addition, by a small oblique band of fibers which cross the upper surface of the

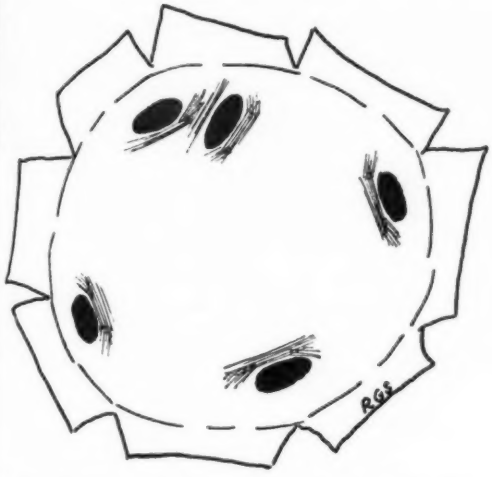

Fig. 3 (Scobee). Drawing showing the inside of Tenon's capsule after the globe has been removed. The points of entrance of all of the oculorotary muscles except the superior oblique are shown as black holes. The thickenings at the posterior margin of each hole are the intracapsular ligaments described by Lockwood.

suspensory ligament." Whitnall[2] is the only investigator who denies the existence of the intracapsular ligaments.

Intermuscular Membrane, Check Ligaments and Muscle Sheaths

It has been previously stated that the middle third of the capsule of Tenon is perforated by the six oculorotary muscles in their course toward their scleral insertions. At the point where each muscle passes through the capsule, the parietal or outer layer is reflected backward along each muscle as a sheath—*the muscle sheath.*

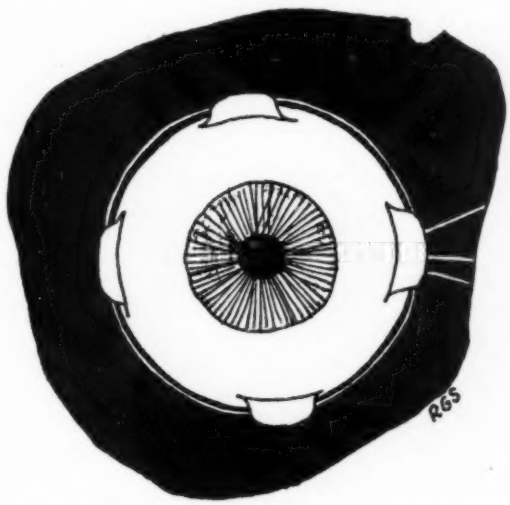

Fig. 4 (Scobee). Diagram showing the intermuscular membrane running between the four rectus muscles. The muscle sheaths are not clearly shown but the three normal check ligaments of the medial rectus muscle are shown fanning out from muscle sheath to periorbita.

According to some investigators, the sheath of each muscle extends all the way back to the point of origin of the muscle, although it is quite true that the sheaths in the posterior half of the orbit become quite thin and difficult to distinguish.

The portion of the fascia of the orbit forming the muscle sheaths is known as the *muscular fascia* and, in addition to ensheathing each muscle completely, the muscular fascia stretches as a membrane between the adjacent margins of the rectus muscles.

This latter structure is known as the *intermuscular membrane* and is usually thickest between the superior and lateral rectus muscles. According to Whitnall[2] the backward extension of the intermuscular membrane is usually greatly exaggerated and practically only extends for a short distance behind the globe.

Intermuscular membrane

Poirier[14] described the intermuscular membrane in some detail in 1912 and stated that it separated the fat of the orbit into two layers, one central and the other peripheral, and controlled to a certain extent the progress of any infiltration which might occur in that region. Whitnall objected to Poirier's work on technical grounds and his idea on the posterior limits of the intermuscular membrane has just been cited.

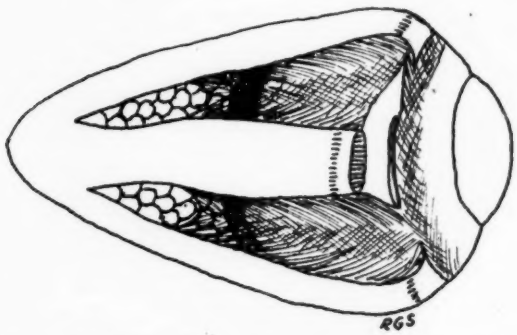

Fig. 5 (Scobee). Drawing showing the method of Jameson in performing what he called "vertical capsulotomy" on the intermuscular membrane. The muscle has been severed from its insertion and is retracted slightly. The intermuscular membrane has been cut vertically from the insertion of the muscle up to the margin of the insertion of the superior rectus muscle.

Butler[15] reported a patient with pus confined to the muscle cone; there were proptosis, pain, and rigors but three incisions for orbital drainage failed to locate any purulent material. It was not until enucleation was performed that pus was encountered and it was found within the muscle cone, confined there by the intermuscular membrane. It is perfectly true that in thin and feeble subjects, however, the membrane may be poorly delimited and difficult to demonstrate.

Jameson[16] was one of the first to mention the clinical significance of the intermuscular membrane in the surgical treatment of heterotropia. In performing his operation of recession, Jameson did what he called a "vertical capsulotomy" in which he incised the intermuscular membrane vertically from the superior margin of the medial rectus at its insertion to the superior rectus and from the inferior margin of the medial rectus downward to the inferior rectus.

Lancaster[17] pointed out that when a muscle's tendon is cut at its insertion into the sclera, the muscle and tendon are not left free and unattached to retract indefinitely into the orbit. On the contrary, the cut tendon retracts only from 3 to 5 mm. or even less because the capsule of Tenon which envelops the muscle is attached to the sclera at about the same distance from the cornea as the tendon insertion, and its connection with the muscle (that is, the intermuscular membrane) is sufficiently firm and intimate to serve as a secondary attachment for the muscle.

If the intermuscular membrane is not divided, one gets nowhere near the maximum effect possible from recession of a muscle. The idea of the recession operation is to reduce the mechanical advantage of a muscle by reinserting it posteriorly on the globe and thus reducing not only its tension but also its arc of contact. If there are broad secondary attachments of the muscle, the mere recession of its tendinous insertion alone, leaving the intermuscular membrane intact, will accomplish but little in comparison with what the operation might do potentially.

The direction in which the intermuscular membrane is cut in the recession operation has a definite bearing upon the amount of correction which may be obtained.[1] As has been mentioned, Jameson sectioned the intermuscular membrane vertically. Even more correction will be obtained if the membrane is sectioned parallel to and within a millimeter or two of the borders of the muscle being recessed, and the membrane should be divided as far posteriorly as it extends.

The intermuscular membrane may have an anomalous development in certain parts which seems to play a definite role in the etiology of heterotropia.[1]

For example, in the patient with intermittent exotropia, it is not at all an uncommon finding for the intermuscular membrane between the lateral rectus and inferior rectus muscles to be markedly thickened and very tough; such an anomaly has the effect of giving the lateral rectus a much wider and heavier attachment than is normal and increasing the action of the muscle to that extent. It is extremely important to divide this thickened portion of the intermuscular membrane completely from the lateral rectus muscle when operating on patients with intermittent exotropia.

The muscle sheaths of all six of the oculorotary muscles have offshoots from their outer or orbital surfaces which become attached either directly or indirectly to the periobita, or to some other portion of the fascia of the orbit. These extensions of the muscle sheaths are known as *check ligaments,* but have also been called *fascienzipfel* or *ailerons.*

Check Ligaments

It is interesting that Tenon[7] believed that the check ligaments altered the direction of the muscles to the extent that, during their contraction, they would not exert pressure upon the globe. Cruveilhier[18] raised doubts about this point, and Lockwood[5] pointed out, quite logically, that the check ligaments of the horizontal rectus muscles, for example, leave the wall of the orbit almost exactly opposite the equator of the globe and run backward nearly half an inch before becoming continuous with the muscle sheaths; this union is far behind the place where the arc of contact of the muscles begins so that the check ligaments would be quite inefficient in preventing pressure upon any part of the globe during contraction of the muscle from whose sheath they arise.

All investigators beginning with Tenon have unanimously agreed on the primary action of the check ligaments in preventing or checking undue contraction of the muscle from whose sheath they spring.

A point that does not seem to be recognized at all, however, is that the check ligaments may act to check motion in *two* directions,[19, 1] particularly if they are unusually thick or have some other anomalous character.

Not only may the check ligaments of the medial rectus muscle prevent excessive contraction of that muscle and thus prevent excessive adduction of the eye, but abnormalities of the check ligaments of this same muscle may also prevent its free relaxation when its antagonist, the lateral rectus, is contracting and thus prevent free abduction of the globe as well. Limitation of motion thus produced may well be an important underlying anatomic factor in the etiology of heterotropia.[1]

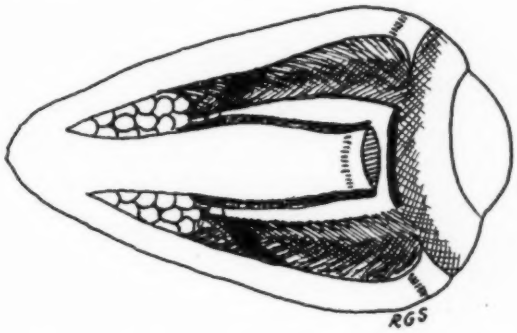

Fig. 6 (Scobee). The intermuscular membrane severed horizontally from the muscle insertion to the posterior extremity of the membrane. It has been divided parallel to the borders of the muscle above and below at a point between 1 and 2 mm. from the muscle borders. This procedure allows a recession to gain its maximum effect.

There are three main types of anomalous check ligaments which are found very commonly in heterotropia:[1] (1) Extra and thickened check ligaments, (2) posterior check ligaments, and (3) fused check ligaments. Any of these types may prevent free abduction of an eye when present on the medial rectus muscle, or free adduction when on the lateral rectus muscle. They must be sectioned at operation if the surgical therapy of heterotropia is to be consistently successful and if the surgeon is to

get the maximum effect desired from the operation of recession.

The check ligaments of the horizontal rectus muscles are not a single simple sheet of fascia as was once thought, but instead there are several sheets fanning out from the muscle sheath to reach insertions in the periorbita. When the check ligaments of the medial rectus, for example, are put on a stretch at the time of surgery, they remind one of the pages of a book fanning out from the binding when the covers of the book are purposely held open with back to back.[19]

The check ligaments of the lateral rectus muscle are more easily demonstrated than those of the medial rectus. They form a wedge-shaped structure with the base of the wedge fastened by a vertical attachment to the periorbita over the malar bone, dorsal to the lateral palpebral ligament, in an area about 6 or 7 mm. long; the apex of the wedge is continuous with the sheath of the lateral rectus.[20]

The greatest width of the check ligaments anteroposteriorly of the lateral rectus is between 6 and 8 mm. while the distance from the anterior end of their orbital insertion to their point of union with the sheath of the muscle posteriorly measures 18 to 20 mm. The point of greatest thickness lies at a distance of from 3 to 6 mm. from the orbital insertion.

These check ligaments are not particularly dense but are composed of a number of bundles of parallel fibers; the fasciculi are separated by small adipose tissue masses, little veins, and lobules of the lacrimal gland. The tiny veins just mentioned are the source of the relatively profuse bleeding encountered in cutting the check ligaments of this muscle when the surgeon has been careful to avoid cutting the anterior ciliary artery until it has been compressed in the suture placed in the tendon of this muscle.

In this posterior two thirds, the check ligaments of the lateral rectus correspond closely with the structure of the muscle

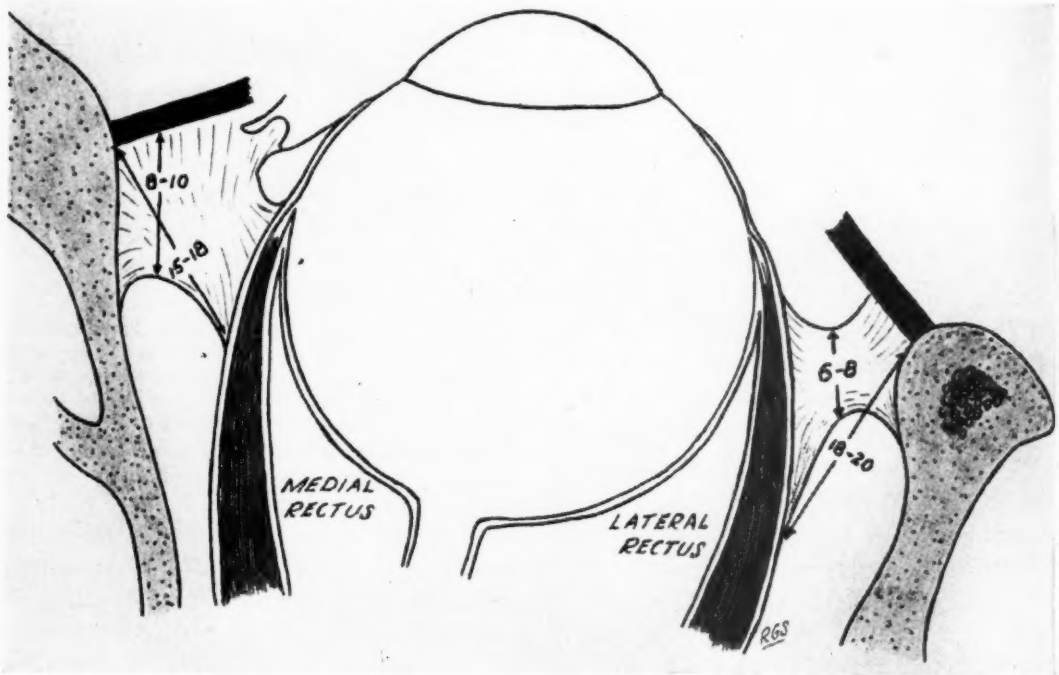

Fig. 7 (Scobee). Diagram showing dimensions of the check ligaments of the horizontal rectus muscles as seen from above.

sheath, while their anterior third often contains plain muscle fibers which have been repeatedly demonstrated by many investigators[20] since Sappey[4] first described them. Most of the fibers of the check ligaments of the lateral rectus are elastic tissue.[5]

The check ligaments of the lateral rectus muscle, in addition to their insertion over the orbital tubercle on the lateral wall of the orbit, are also attached to the posterior face of the lateral palpebral ligament (which is also inserted into the orbital tubercle) and into the conjunctiva of the lateral fornix.

The check ligaments of the medial rectus muscle present a similar triangular formation to those of the lateral rectus[20] and are attached to the medial wall of the orbit over the lacrimal bone just behind the posterior lacrimal crest. They are also attached to the septum orbitale behind the pars lacrimalis muscle of Müller, to the lacrimal caruncle, and to the conjunctiva in the medial fornix.

These check ligaments are wider than those of the lateral rectus muscle (8 to 10 mm. as compared with 6 to 8 mm.), but their thickness of 1.0 to 1.5 mm. is less than that of the ligaments of the lateral rectus. They are shorter (15 to 18 mm.) than those of the lateral rectus (18 to 20 mm.).

Sutton[20] described many hairlike fibrous projections from the check ligaments of the medial rectus which unite with the capsules of the adipose masses in that region. There are no interstices similar to those found in the lateral rectus check ligaments. Plain muscle fibers have also been demonstrated in the anterior third of these check ligaments. According to Motais, the check ligaments of the medial rectus have more elastic fibers than do those of the lateral rectus.

Any of the anomalies of the check ligaments previously mentioned may play an important role in the etiology of heterotropia. It is, therefore, essential that the check ligaments be divided from the muscle sheath when a recession operation is done if the surgeon is to get the full and proper effect from this procedure.[19, 1]

The fact that the check ligaments of the medial rectus muscles are attached to the caruncle, to the medial conjunctival fornix, to the posterior surface of the medial palpebral ligament, and to the periorbita of the medial wall of the orbit has been mentioned. If the full effect of a recession operation is to be gained, all of the check ligaments must be severed.

In the past, various writers have strenuously objected to cutting the check ligaments on the grounds that in the case of the medial rectus such a procedure leads to retraction of the caruncle. Quite the opposite is true and for obvious mechanical reasons.[1] The check ligaments prevent excessive contraction of the muscle from whose sheath they arise; this is to say that they prevent undue shortening of the muscle.

A recession operation is the equivalent in one respect to a shortening of the muscle in contraction in that either event places a certain amount of stress upon intact check ligaments. When the check ligaments of the medial rectus muscle are put under stress, since they are attached to the caruncle, there results a retraction of the caruncle. If, however, the check ligaments of the muscle are divided before the muscle is recessed, no strain is placed upon the check ligaments and hence there is no retraction of the caruncle.

The facts speak for themselves and are proved by a series of over 120 patients[1] with esotropia in whom the check ligaments of the medial rectus muscle have been completely severed from the sheath of the muscle prior to a recession of that muscle; there has been not a single instance of retraction of the caruncle postoperatively in the entire series.

A word of caution must be inserted about exercising care when cutting check ligaments from the muscle sheath. This procedure does not involve stripping the sheath from the muscle to leave the latter bare and exposed; it merely entails cutting the check ligaments close to the point where they origi-

nate from the sheath. There are serious objections to stripping a muscle of its sheath because adhesions invariably form between bare muscle and any adjacent tissue and this is very undesirable.

Ledouble[21] first described a fasciculus of striated muscle in the check ligaments of both horizontal rectus muscles and said that they were fairly frequent in occurrence and were presumably offshoots of the muscles themselves. Such offshoots are not uncommon in patients with heterotropia and may play a role in the etiology of the heterotropia.[1]

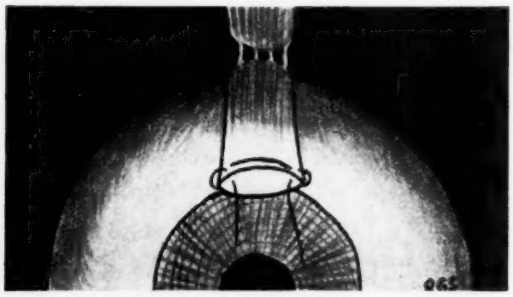

Fig. 8 (Scobee). Schematic drawing of the eyeball showing only the superior rectus severed from its insertion and pulled forward and downward by means of sutures. The levator is shown above and the usual four dentate connections between the two muscles can be seen put on a stretch by this maneuver.

Muscle sheaths

The relations of the sheaths of the rectus muscles are all of the same type; at the point where muscle tendon is inserted into sclera, the sheath passes from the internal or ocular surface of the muscle backward as part of the middle third of the capsule of Tenon. The sheath passes from the external or orbital surface forward to find attachment partly into the fornix of the conjunctiva and partly into the sclera near the limbus. There are certain modifications in the case of each individual rectus muscle. The relations of the superior rectus, for example, are complicated by the presence of the levator.

The muscle sheath of the superior rectus is similar to those of the other recti already mentioned but it has no check ligaments similar to those of the horizontal rectus muscles. Its sheath and that of the levator are rather intimately attached since the two muscles are in close apposition throughout most of their course.

Through its adherence to the levator via their sheaths, the superior rectus gains a form of attachment to the aponeurosis of the levator and to the superior tarsal cartilage in addition to its attachment to the bulbar conjunctiva and the superior conjunctival fornix.

The lateral attachments of the levator to the orbital walls act as check ligaments for that muscle and, according to Cruveilhier,[18] make the tarsal cartilage of the upper lid function like the visor of a helmet. The check ligaments of the levator also function as check ligaments of the superior rectus because of the intimate relationship between the two muscles and furthermore the superior rectus can partially lift the upper lid for the same reason.

If one carefully pulls the superior rectus downward away from the levator, there are usually four fairly thin sheets of fascia running longitudinally between the two muscles, and these sheets, when put on a stretch and viewed anteriorly, have a dentate appearance.

If a recession operation on the superior rectus is to be fully effective, not only must the intermuscular membrane be divided on each side of the muscle as far back as it extends, but the connections between the sheaths of the superior rectus and the levator must be gently divided without stripping either muscle of its sheath and leaving it bare.

If this latter procedure is not done, the effect of a recession will be minimal because the attachments between the superior rectus and the levator will prevent the recessed superior rectus from retracting properly.

Failure to observe this simple precaution is responsible for much of the widespread dissatisfaction with recession of the superior rectus muscle on the grounds that it is a relatively ineffective and always unpredictable procedure. When the operation is properly done, it is neither ineffective nor unpredictable.

The superior oblique in early fetal life has no connection with the wall of the orbit. As the muscle develops, it gradually approaches the wall of the orbit and finally becomes attached to it. In the course of the development, the trochlea is a secondary structure. The attachment of the sheath of the superior oblique to the trochlea is by small sheets of fascia given off both before and after the sheath containing the muscle has passed through the trochlea.[20]

Various authorities disagree about the extent of the muscle sheath of the superior oblique. Lockwood[5] believed that it extended only from Tenon's capsule to the trochlea. Sutton[20] found it extending on backward through the trochlea but did not state how far. Fink[22] found that a recognizable sheath extended some 8 to 10 mm. posteriorly from the trochlea.

Others have stated that it extends about half way backward from the trochlea to the origin of the muscle, while still others claim that it covers the muscle in its entire course from origin to trochlea and from trochlea to the globe. It seems likely that the thick and obvious portion of the sheath runs from the capsule to and through the trochlea and then becomes progressively thinner as it is traced posteriorly until it blends almost imperceptibly with the perimysium.

Berke[23] has effectively demonstrated that the tendon of the superior oblique is intimately attached to its encasing sheath from trochlea to globe by countless tiny trabeculae. If the tendon is divided within its sheath, the cut ends retract a short distance before they are stopped by the anchoring effect of these hundreds of tiny trabeculae.

Because of this fact, Berke has proposed the very effective procedure of intrasheath tenotomy of the superior oblique as a lengthening operation for that muscle in patients in whom it is desired to lessen its action. Gradations of effect may be obtained from the smallest, by simple tenotomy, to the greatest, by actual tenectomy of varying lengths of tendon. The operation is easy to do and is very satisfactory.

The sheath of the inferior rectus is quite similar to that of the other rectus muscles except anteriorly where it divides into two layers: a superior one which joins the capsule of the globe and runs forward to become inserted into the sclera near the limbus, and an inferior layer which is inserted into the posterior border of the sheath of the inferior oblique as well as into the suspensory ligament of Lockwood.

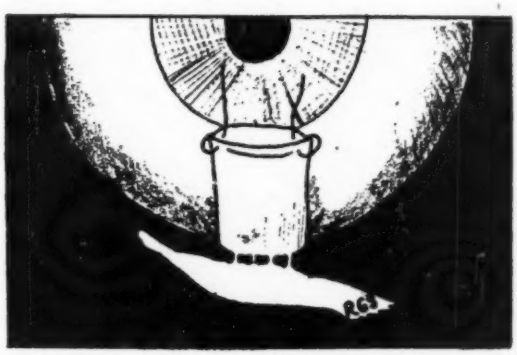

Fig. 9 (Scobee). Schematic drawing of the eyeball showing only the inferior rectus severed from its insertion and pulled forward and upward out of its bed in the suspensory ligament by means of traction sutures. The inferior oblique is represented below and the usual four dentate connections between the inferior rectus and its bed can be seen when put on a stretch by this maneuver.

Once the inferior rectus has passed through its opening in the capsule of Tenon, it runs forward to its insertion lying in a bed formed by the suspensory ligament. If the inferior rectus is lifted carefully upward and out of its bed, it is usual to find four thin sheets of fascia running longitudinally with the muscle and connecting the muscle

sheath to the suspensory ligament; viewed anteriorly when put on a stretch, these have a dentate appearance similar to the same connections found between the superior rectus and the levator.

It is to be emphasized that the sheath of the inferior rectus is attached by fascial sheets only to the suspensory ligament of Lockwood and to the posterior surface of the sheath of the inferior oblique. The bands to these two attachments are the check ligaments of the inferior rectus. Unless they are divided, the recession operation is neither completely successful nor satisfactory.

For many years, one objection to recession of the inferior rectus has been that the procedure leads to retraction or drooping of the lower eyelid. It will be noted that the sheath of the inferior rectus has no direct connection to the tarsus of the lower lid. As will be pointed out in subsequent paragraphs, however, the suspensory ligament of Lockwood is connected to the tarsus of the lower lid and so is the check ligament of the inferior oblique. Thus, the sheath of the inferior rectus has two indirect connections with the lower lid.

If the inferior rectus is recessed and the connections of its sheath with the sheath of the inferior oblique and with the suspensory ligament of Lockwood are not divided, backward tension is placed on both of these structures—tension which is transmitted through them to the tarsus of the lower lid, resulting in retraction or drooping of that lid.

If, however, the sheath of the inferior rectus is divided from its attachments in the operation of recession, no backward tension is created and there is no drooping or retraction of the lower lid.

Again, as in the case of retraction of the caruncle, the simple mechanical facts are obvious and have been proved in a suitable number of cases. Furthermore, unless these attachments of the sheath of the inferior rectus are divided in the operation of recession, the operation will not have its maximum effect and the results will be highly unpredictable, just as in the case of the superior rectus.

As soon as the surgeon stops avoiding the vertical rectus muscles because recession operations on them are "unsatisfactory" and begins to do the operation properly with due regard for the mechanics of the situation, just so much sooner will the surgical therapy of the vertical deviations gain a sound footing on the basis of cases successfully handled.

Ligament of Lockwood

Before considering the sheath and check ligament of the inferior oblique, it would be well to mention the ligament of Lockwood. We shall return to the original work of Lockwood[5] for his own description first. "The suspensory ligament is a band of fibrous tissue stretched, like a sling, from one side of the orbit to the other. The fibers which compose it converge at each end to be inserted into the malar and lacrimal bones; in the middle they diverge to form a shallow cup upon which the eye rests. The widest part of the suspensory ligament is intimately woven with the capsule of Tenon, but not to such an extent as to conceal the identity of its fibers."

Lockwood continued: "The inferior portion of the capsule of Tenon is thickened and has an aperture through which the inferior rectus passes. The posterior part of Tenon's capsule inferiorly is thickest just behind this opening for the inferior rectus and gradually becomes thinner as it passes round the eye; the anterior part looks crescentic and sends a long thin horn backward beneath the inferior rectus, forming the upper layer of the underpart of its sheath, and another forward to become continuous with the ocular conjunctiva and the inferior tarsal cartilage. It is united to the inferior cartilage by a short thick process which it gives off in front. In contact with its lower surface is the sheath of the inferior oblique.

Where the suspensory ligament mingles with the capsule of Tenon, the thickness is slightly more than one tenth of an inch. The most important action of the suspensory ligament is, obviously, to support the eye."

Fink[24] has recently emphasized and carried forward the work of Lockwood. He has shown that both inferior rectus and inferior oblique have attachments of their sheaths to the suspensory ligament which vary tremendously in density and number from one specimen to the next. Fink makes the flat statement that ". . . the inferior rectus apparently has no direct attachment to the orbital septum or margin of the orbit"; Lockwood, on the other hand, made no such statement although he strongly implied that this was the case by merely not mentioning any attachment of the inferior rectus to the orbital wall.

The sheath of the inferior oblique extends from its origin on the floor of the orbit along the muscle to a point where its outer layer fuses rather firmly with the inferior surface of the ligament of Lockwood. The muscle itself, accompanied by the inner layer of its sheath, meanwhile penetrates the suspensory ligament and the capsule of Tenon at a point somewhat posterior and lateral to the site of penetration of the inferior rectus muscle. The inferior oblique passes on through the suspensory ligament and the capsule of Tenon to its insertion near the macula.

Near the insertion of the inferior oblique, its sheath spreads out anteriorly to fuse with the sheath of the lateral rectus muscle; posteriorly, its sheath spreads out to fuse with the dura of the optic nerve.[22] The union between the sheaths of the inferior oblique and lateral rectus is sometimes quite firm and effectively binds the two muscles together while at other times it is very loose.[24]

The posterior extension of the sheath of the inferior oblique usually fuses with the dura of the optic nerve and is pierced by the posterior ciliary arteries and nerves which enter the globe temporal to the optic nerve. Fink[22] has emphasized that effective surgery of the inferior oblique includes division of both of the attachments of its sheath to the lateral rectus muscle and to the optic nerve and a good deal of caution is required in separating the latter.

Motais[25] originally described what he considered to be a check ligament for the inferior oblique, but Whitnall[2] denied the existence of any such structure. The recent work of Fink[24] confirms the general findings of Motais, however, and is detailed enough to leave little doubt that such a structure exists.

According to Fink, the check ligament of the inferior oblique muscle leaves the anterior border of that muscle about 8 or 10 mm. from its point of origin and courses obliquely forward, outward, and downward; it leaves the sheath of the inferior oblique at an angle of about 110 degrees.

The check ligament is about 10 to 12 mm. in length and is probably better described as a broad meshwork of fascial fibers rather than as a definite band or ligament, but it serves as an attachment for the sheath of the inferior oblique to the floor of the orbit at a point 4 or 5 mm. behind the orbital margin. Its width varies at different parts of its course so that it presents the shape of two triangles united by their apexes.

Together with the inferior oblique itself, the check ligament forms a kind of musculoaponeurotic loop, the two ends of which are inserted near the orbital margin, one at the outer angle and the other at the inner angle. Strands from the sheath of the inferior rectus, which might be considered as its check ligaments, embrace the midde portion of this loop so that when the inferior rectus contracts, these attachments stretch the loop.[24]

Motais[25] called the ligament of the inferior oblique a ". . . pulley of reflection," suggesting that when the muscle contracted, its check ligament pulled the middle of the belly outward and laterally, thus making its

line of traction on the globe a little less oblique. This would, of course, result in greater elevation and less extortion, particularly in adduction, and would explain even more fully the observed phenomenon of normal overaction of the inferior oblique.[19]

The septum orbitale

The septum orbitale is another portion of the fascia of the orbit which is a connective and elastic tissue membrane extending from the arcus marginale all around the opening of the orbit centrally toward the palpebral fissure. The septum in the upper lid is inserted into the skin of the upper lid, being carried forward by the levator fibers.

Whitnall[2] has pointed out that when the downward extending septum meets the levator fibers, its deeper layers are carried slightly backward to fuse with the levator's aponeurosis, thus shutting off the orbital contents from the interior of the upper lid.

He has also shown that in the upper lid, the septum orbitale is not fixed in a vertical plane but moves and folds with movements of the lids. The septum orbitale coming from the inferior portions of the arcus marginale is inserted into the inferior margin of the tarsal plate of the lower lid.

The palpebral ligaments

The medial and lateral palpebral ligaments are the final portions of the fascia of the orbit to be considered. They arise as extensions of the ends of the tarsal plates in the upper and lower lids and fuse at the inner and outer canthi to run as single ligaments to the medial or lateral wall of the orbit, as the case may be.

Whitnall[2] has shown that the medial palpebral ligament runs anterior to the dome of the lacrimal sac; immediately after it is formed, however, by a confluence of extensions from the medial ends of the tarsal plates, it gives off a smaller posterior division which runs behind the dome of the lacrimal sac to be inserted on the posterior lacrimal crest and it is to this posterior extension that the check ligaments of the medial rectus muscle become attached.

The lateral palpebral ligament is attached to the orbital tubercle of the zygomatic bone. Injuries which result in complete division of the medial palpebral ligament are quite disfiguring cosmetically because of the sunken appearance of the medial canthus which is produced.

Callahan[26] has recently reported the use of fine steel wire passed from the cut end of such a sectioned medial ligament to the medial wall of the orbit and through small holes drilled in the bone there by means of a dental drill; this results in a very satisfactory repair by abolishing the sunken inner canthus.

640 South Kingshighway (10).

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THE VERTICAL ELEMENT IN THE CAUSATION OF SO-CALLED HORIZONTAL CONCOMITANT STRABISMUS*

Surgical principles for the vertical corrections

Edmund B. Spaeth, M.D.
Philadelphia, Pennsylvania

Strabismus is an abnormality of the visual axes due to several different etiologic conditions. At this time paralytic lateral strabismus is not being considered but all other forms have a definite relationship to this presentation.

It seems, if one is to speak of a lateral concomitant squint with a vertical component present in that squint, that that case is not purely concomitant. If this is true, then also is it true that the most common form of pure lateral concomitant strabismus is the classical alternating type of strabismus wherein the patient has the same degree or angle of squint for near and for distance, both objective and subjective angles being the same, the refractive error being practically equal and negligible in each eye, and this having no relationship to the degree of squint in any convergence-accommodation ratio relationship. In this type of strabismus the patient frequently uses each eye at will, or one eye for near and the other eye for distance, and in these cases there is alternating suppression with alternate functional central scotomas. It is rather interesting to compare this type of squint (in considering vertical relationship) with a classical case of accommodative strabismus, that specific case also having a vertical component.

It is likely that the greatest etiologic factor in the development of strabismus is in the sensory realm. There is no doubt, however, that a large number of cases have, as a most important factor in etiology, a motor defect. It makes little difference whether this is peripheral (orbital) or central—that is, nuclear. See Figure 1. This patient, with a paralysis of the inferior oblique muscle and the superior rectus muscle, both on the left, could hardly have a peripheral incomplete abnormality. When one adds to that the finding of a unilateral ptosis on the right, then also does it seem certain that her condition must have been a bilateral, central, brainstem (probably internuclear) congenital developmental defect.

Figure 2 shows a frank alternating strabismus with no vertical component whatsoever; the right eye fixing, or the left eye fixing; it also illustrates the results following surgery. Even in this, however, with

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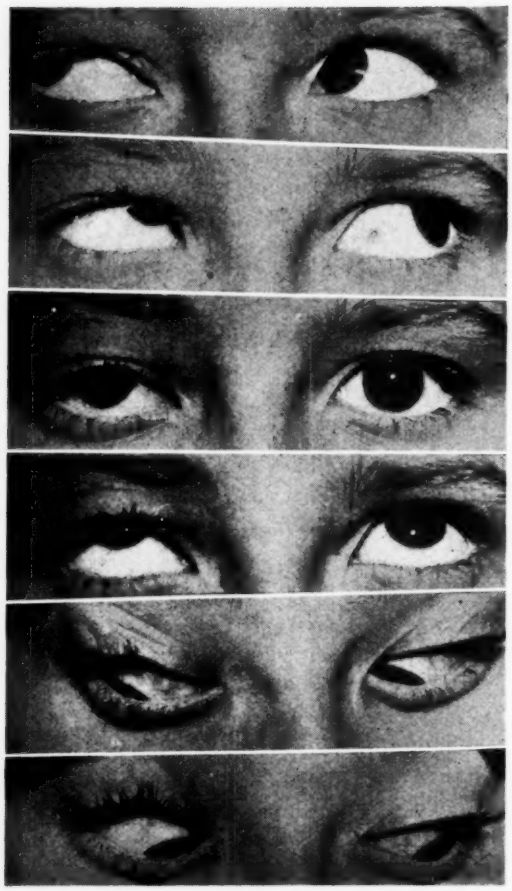

Fig. 1 (Spaeth). Paralysis of the inferior oblique and superior rectus muscles, both on the left. This could hardly have a peripheral incomplete abnormality.

careful study one can see a difference in the palpebral fissures and a bare suggestion of retraction in the right eye; that is, a motor defect. The patient had 6/6 vision in each eye; there were no limitations in ocular motility; he had a negligible equal refractive error of less than plus one (hyperopia), sphere and cylinder combined, bilateral suppression scotomas, and diplopia could not be elicited at any time.

Figure 3 illustrates an almost pure accommodative squint. Vision was 6/12 and 6/6. The refractive error was a plus four, total sphere and cylinder combined, in the right; and a plus two, sphere and cylinder combined, in the left eye. The squint increased at near and disappeared almost wholly under full cycloplegia and with the correcting glasses. There was no doubt that this case had a marked so-called accommodation-convergence relationship, without any lateral oculomotor palsy. The patient showed, however, in right lateral rotations, a definite overaction of the left inferior oblique muscle, the barest amount of overaction of the right inferior oblique muscle, and a slight underaction of both superior oblique muscles. There was no certainty that this child did not have a very moderate elevator palsy of some type. The case was wholly free from strabismus at near, under cycloplegia, and with her full correction.

If a paralysis of a vertical elevator was absent, then this case of practically pure accommodative strabismus must have had, in addition, an accompanying overaction of the inferior oblique muscles. One wonders whether this could be connected in any way with her overconvergence, in this way causing a bilateral inferior oblique spasm. That was simultaneous with the overconvergence and became most manifest when the eye was in a position for the maximum of vertical inferior oblique muscle action; that is, the inferior oblique muscle spasm being at maximum in the left eye with right rotations, and in the right eye with left rotations.

The question of a vertical component as a part of convergent strabismus, in fact of almost any type of lateral strabismus, should give us an inkling as to the causation of strabismus in a certain percentage of cases. This seems especially so when one studies all cases of strabismus, in detail, with the screen test, fixational reflexes, and with prisms in the six cardinal positions. Very few cases of apparently concomitant, nonaccommodative (even of accommodative), monocular lateral squint are found which do not have disturbances in the vertical rotations. The question of cause and effect for each of the two components is at least interesting.

Binocular vision is achieved by the de-

velopment of a conditioned reflex and not because of the presence of an inherent urge that compels the infant or young child to swing into single binocular vision, if this is possible. Fusion as such follows the establishment of this as a conditioned reflex. It (the reflex) is the result of the search and struggle for single binocular vision and the fixation of that, when achieved, and not because of it; that is, fusion or single binocular vision.

Fusion, as a term for an ocular function, is better considered as being quantitative in character, rather than qualitative. The simultaneous use of both eyes, and with this a single cerebral visual concept having certain characteristics (single binocular vision),

Fig. 2 (Spaeth). A frank alternating strabismus with no vertical component whatsoever.

while clumsy as a definition is a qualitative characteristic. The degree of perfection in this function (single binocular vision) as a conditioned reflex is to be measured in terms of fusion.

There are two factors which permit the formation of a perfectly conditioned reflex wherein the child obtains a single cerebral visual concept. They are the synchronous use

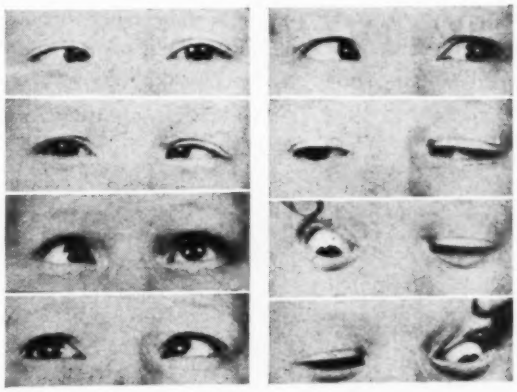

Fig. 3 (Spaeth). An almost pure accommodative squint.

of the two cerebral hemispheres and, at the same time, the simultaneous use of the two eyes. This, of necessity, implies the presence of two attributes or essentials; one is in the motor sphere, the other is in the sensory realm. If there are neither sensory nor motor obstacles present, the patient should progress normally to single binocular vision and this should be well established by the 4th or 5th year of life.

Motor obstacles may include such conditions as temporary to permanent anatomic disturbances from birth injuries, defects affecting the orbits and the globes, faulty insertion of muscles, fibrosis of a muscle (as one sees in the Duane syndrome) which is probably an imperfect mesodermal development of an ocular muscle, aplasia of an oculomotor nucleus, and contracture of the antagonist of a muscle imperfectly exercised.

Sensory obstacles must include not only the many possible peripheral sensory retinal situations, but also possible conflicts between the occipital lobe involuntary accommodational reflex, the frontal lobe voluntary accommodation-convergence reflex, and the internuclear brain stem accommodation-convergence reflex—normally of greatest importance in conditioning binocular single vision. An abnormal convergence innervation consequent to disturbances in these three is probably the greatest of the faults in the sensory realm. Such defects are either an

excess in convergence or a deficiency in convergence. A discussion of these is not relevant at this time, except to take recognition of the role they play. Also, one must not

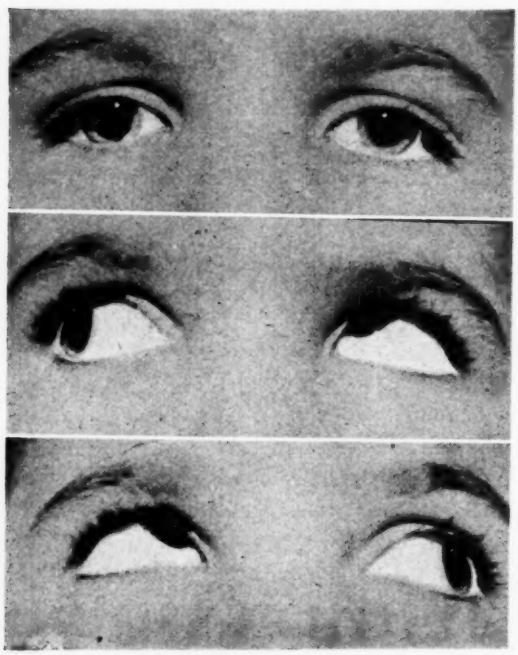

Fig. 4 (Spaeth). A case of alternating divergent strabismus of a moderate degree of severity, with marked inferior oblique muscle overaction.

overlook the fact that the convergence excess of accommodative squint, with full correction (refraction), frequently shows a convergence insufficiency.

As the infant strives to develop single binocular vision, as a result of his awareness to diplopia, he may fail in meeting and overcoming sensory or motor obstacles and so he also fails to obtain normal single binocular vision. Instead, strabismus of some type develops through the conflict between a normal sensory situation and a faulty motor balance; or a faulty sensory state and a normal motor condition; or the not uncommon condition wherein both are faulty although to dissimilar degrees.

Faulty vertical function is an outstanding motor obstacle. It is not necessarily limited to convergent strabismus but it is for some reason somewhat less common in divergent strabismus. Divergent strabismus, except the monocular type, is not an attribute of very early childhood, and the incidence of divergent squint is rare, as compared with convergent squint. One might suspect that diverging visual axes discourages an overaction of the oblique muscles. This, however, seems to reverse the order of cause and effect. Disturbances in the vertical element nevertheless do appear frequently.

Figure 4 is a case of alternating divergent strabismus of a moderate degree of severity. The child in the upper photograph is fixing with the left eye. (See position of the pupillary reflexes.) Movements up and to the right and up and to the left show a marked overaction of the contralateral inferior oblique muscle. There were no disturbances

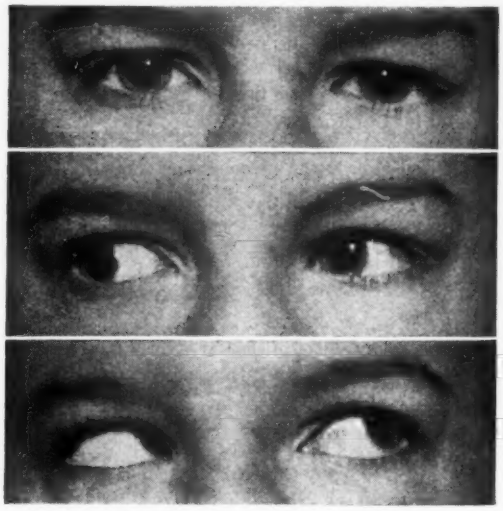

Fig. 5 (Spaeth). A unilateral case in which the inferior oblique muscle is rotating normally in amplitude but its conjugate yoke muscle is rotating to a limited degree.

in visual acuity. This is the type of case which will certainly continue to advance into more and more pathologic divergence.

In this paper, the term "overaction" is being used rather loosely. It is not always applied upon pure physiologic principles, but only in terms of an increase in the amplitude of excursions of the eyeball as the re-

sult of the action of a certain muscle. In addition, inhibitional palsy is not being considered for it is not relevant here.*

The vertical component is not necessarily the result of the overaction of an inferior oblique muscle. There is no doubt that in many instances one can state definitely that there is limitation in the field of action of a superior rectus muscle and, therefore, the contralateral inferior oblique muscle is overacting secondarily. This can be explained upon the basis of two possibilities.

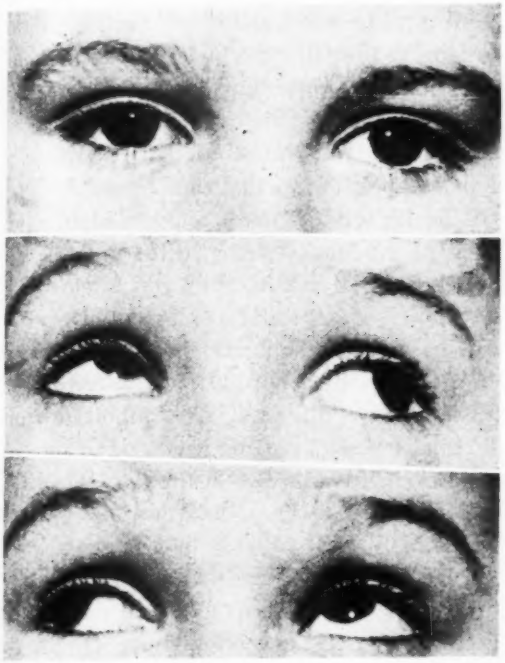

Fig. 6 (Spaeth). A bilateral case of the inferior oblique muscle rotating normally in amplitude but its conjugate yoke muscle rotating to a limited degree.

The first of the two is that the inferior oblique muscle is rotating normally in amplitude but its conjugative yoke muscle is rotating to a limited degree. The action of the inferior oblique muscle is relatively greater now only because of its normal nerve and muscle anatomy. This would be purely a peripheral situation and would exclude any effect from a desire for binocular single vision. Figure 5 seems to be such a case, unilaterally, and Figure 6 bilaterally. Neither of these patients had any lateral strabismus when under the cycloplegia, as photographed.

Fig. 7 (Spaeth). A true overaction is caused when a greater than normal innervational effort is put forth in the attempt to obtain a maximum amplitude of movement in a paretic muscle, causing overstimulation of the conjugate yoke muscle.

Fig. 8 (Spaeth). A relative underaction of the right inferior rectus muscle and an overaction of the left superior oblique muscle. A congenital ptosis with a possible involvement of the superior rectus muscle is also present.

The second likely and certainly more frequent situation, as seen, for instance, with a paretic superior rectus muscle, occurs as follows. A greater than normal innervational effort is put forth by the patient in his attempt (or wish) to obtain a maximum amplitude of movement in this paretic

*This applies, in the vertical disturbances, in cases in which the patient is using, for fixation, the eye with an involved vertically acting muscle. The inhibitional involvement will then appear in the opposite eye. In such instances, considering the basic purposes of this presentation, both horizontal deviation and vertical inhibition might be seen in the same eye.

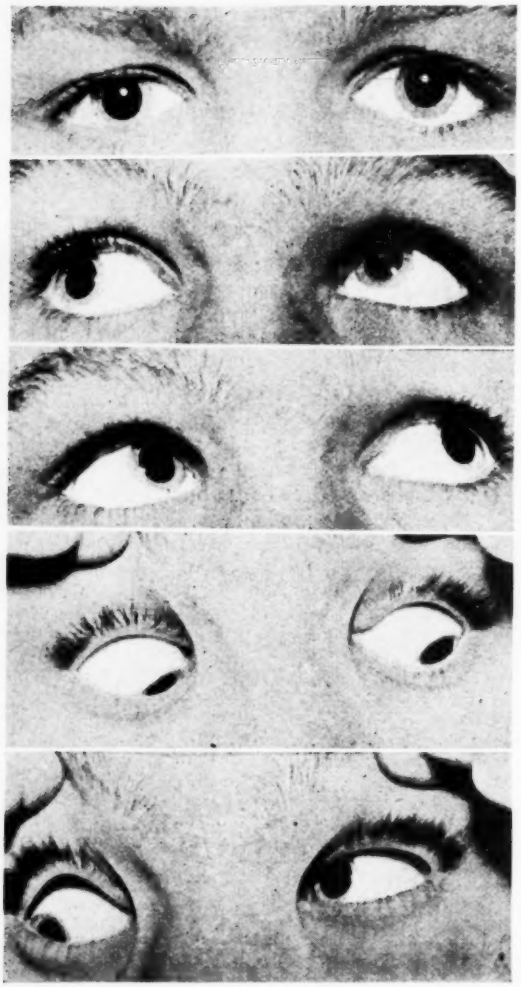

Fig. 9 (Spaeth). An accommodative strabismus, the horizontal component of which was eliminated by cycloplegia.

muscle, and perhaps to hold binocularity (distaste for diplopia) and because of that, the conjugate yoke muscle, cerebrally controlled and from a nuclear source, has a true overstimulation, and thus a true overaction. Figure 7 illustrates this condition and shows especially the overaction in the right eye.

Similarly, one should see this same relationship between an overacting superior oblique muscle in one eye and an underacting inferior rectus muscle in the contralateral eye. In these instances true limitation of the ocular rotations are picked up more rapidly.

Figure 8 shows, in downward movements to the right, a relative underaction of the right inferior rectus muscle and an overaction of the left superior oblique muscle. This was not present in left lateral rotations in which the depressors of both eyes were equal and balanced in the extent of their motility. The comparison of the maximum possible action of each inferior rectus, separately, is the important diagnostic criterion as to laterality in this case.

Figure 8 illustrates another complexity. This complication is that of a congenital ptosis of the left eye with a possible involvement of the left superior rectus muscle. Repeated studies in this child did suggest the possibility of a primary or essential overaction of the left superior oblique muscle in the presence of a normal right inferior rectus muscle. This was based upon a comparison in degrees of the rotations of the two inferior rectus muscles in the two eyes. The patient thus has a possible limitation in the action of the left superior rectus muscle, the left levator muscle, possibly the inferior rectus muscle in the right eye, and an overaction of the superior oblique muscle in the left eye. These muscle details are emphasized to illustrate the complexities in the diagnosis of the primary and the secondary deviations, as well as the effect of unilateral vertical muscle antagonists.

Another possibility which must be seriously considered is not a true or even relative overaction of a conjugate yoke muscle (primary paretic pathologic process in one eye; overaction in the other eye) but the relative overaction of the direct monocular muscle antagonist to a paretic muscle. In considering any one of the vertically acting muscles as, for instance, the superior oblique muscle, the question (fig. 9) is not one of a probable nuclear aplasia of the fourth-nerve nucleus or of any type of possible fourth-nerve involvement; it is specifically the consideration of a frankly underacting superior oblique in one eye with a strongly overacting inferior oblique in that same eye, the eyeballs being rotated for this demon-

stration into the position of greatest vertical action for the oblique muscles under consideration.

The child in Figure 9 has an accommodative strabismus (O.D.), the horizontal component of which was eliminated by cycloplegia (as can be seen in the upper of these photographs) wherein downward rotations to the right showed the paresis of the left superior oblique muscle, while upward rotations to the right brought out the overaction of the inferior oblique muscle of the same eye, the result of a direct monocular antagonist.

Another matter is relevant at this point. It is the all too frequently heard statement of a vertically acting "weak muscle." This adjective can be applicable only in cases of faulty muscle-tendon insertions, or of mesodermal maldevelopment. All other anatomic and hence functioning causes are more likely defects within (and in) innervation; hence, truely paretic in nature.

Figure 10 shows a similar but even more convincing case, although it is not a pure lateral concomitant strabismus. The patient had an almost complete paralysis of the superior division of the left third nerve. Marked secondary overaction of the right inferior oblique muscle is seen, mild underaction is present in the left inferior oblique muscle (perhaps the result of a subtotal paralysis of the left third nerve), and with it an overaction in the two direct monocular antagonists—the inferior rectus on the left, the result of the superior rectus paralysis; and the superior oblique on the left, the result of the underaction of the left inferior oblique muscle.

Figure 11 is a case in which both levators of the left eye were involved; that is, an evident impairment of the left inferior oblique muscle and a lesser degree of impairment of the left superior rectus muscle, suggested as such because of the overaction of the right inferior oblique muscle. It is interesting that in this patient there is no overaction in either of the monocular

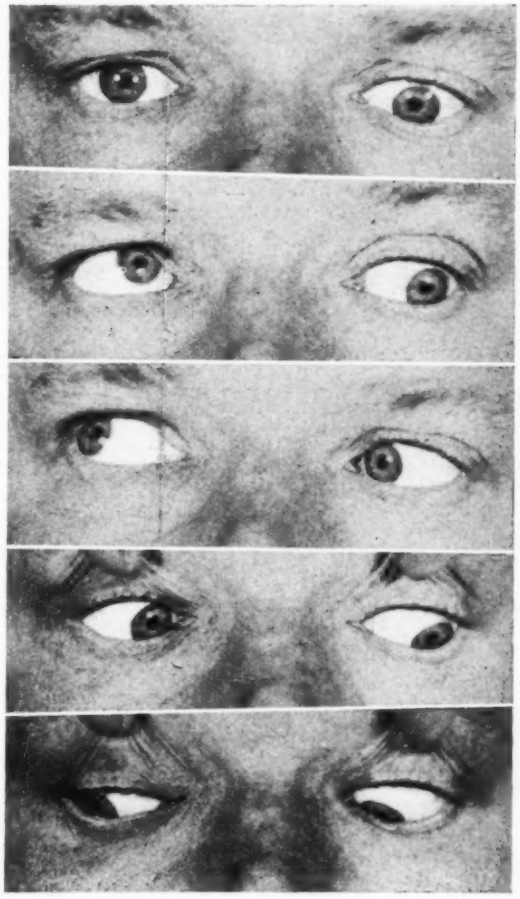

Fig. 10 (Spaeth). An almost complete paralysis of the superior division of the left third nerve.

direct antagonist muscles. This case illustrates well the fallacy of orthoptics in this type of case, except for those exercises to combat the amblyopia. This patient had had many months of exercises in another city, and the mother stated "without changing the cross-eye in any way." One wonders whether the vertical element was considered at the time the ophthalmologist outlined the orthoptic treatment.

Figure 12 presents a mild monocular convergent strabismus in the left eye, with a marked amblyopia. There is a marked increase in the convergent strabismus present when the patient is compelled to use the paretic inferior oblique muscle on the left. There is, however, no gross overaction of

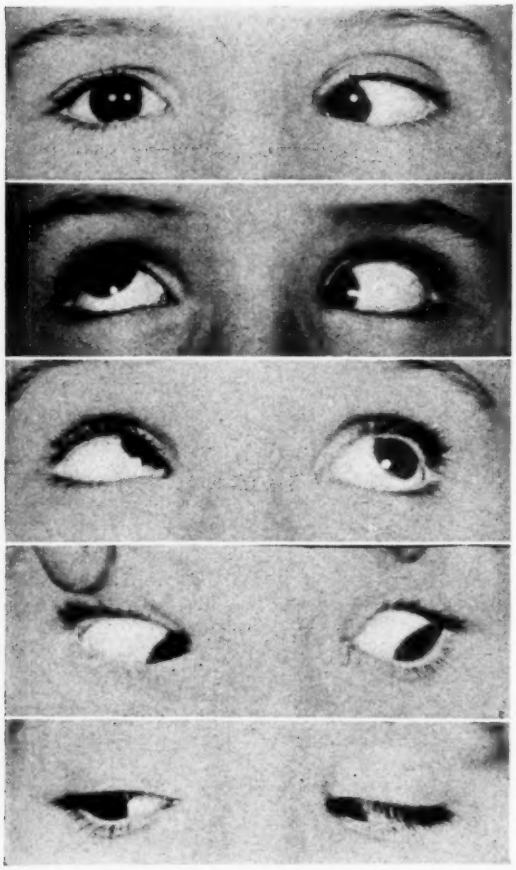

Fig. 11 (Spaeth). Impairment of both elevators of the left eye.

his conjugate muscle; in this instance, the superior rectus muscle on the right. Also, in his case there is little, if any, overaction of the monocular antagonist, the superior oblique muscle of the left eye. His vertical rotations to the opposite left side were normal and balanced. Here, certainly, one can see a *spasm* of an internal rectus muscle consequent to a vertical muscle disturbance.

Figure 13 is a case of bilateral limitation of equal degree in the field of the inferior oblique muscles.

This patient, aged four years, had a transient convergent strabismus, present when fatigued but with some amblyopia already established, showing the sensory suppression put forth for physiologic ocular comfort. There was neither contralateral synergist nor homolateral antagonist muscle overaction. There may have been some underaction of the left superior oblique muscle. The condition illustrated is essentially one of bilateral hypophoria. The patient is suppressing in one eye only; he has good stereopsis; and the inferior oblique muscles are both faulty. Torticollis was present and was constantly toward the right.

The problem of differentiating an overacting inferior rectus muscle with a contralateral relatively (comparative) underacting superior oblique muscle from an underacting superior rectus muscle with a contralateral relatively overacting (again comparative) inferior oblique muscle appears occasionally for solution in any certain case. Both of these conditions, frequently, have a marked degree of lateral strabismus.

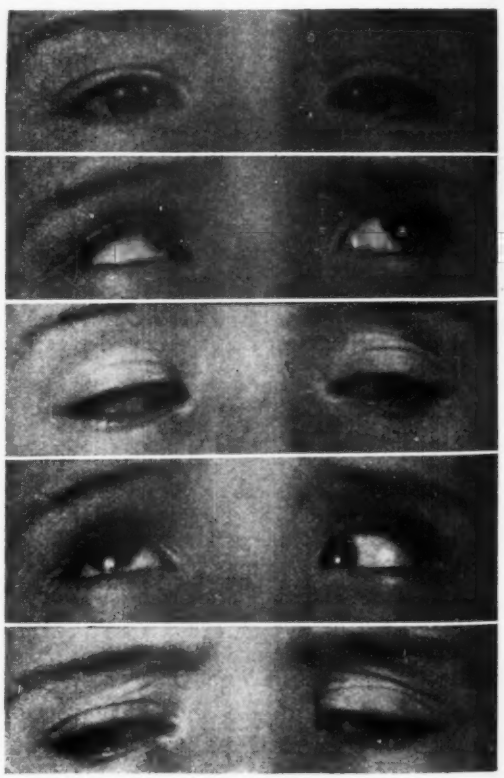

Fig. 12 (Spaeth). A mild monocular convergent strabismus with a marked amblyopia. There is a marked increase in the convergent strabismus present when the patient is compelled to use the paretic inferior oblique muscle.

Figure 14 illustrates this. The child has a monocular convergent strabismus on the left with vertical movements to the right practically equal and balanced. Vertical movements, however, downward and to the left show an overaction in the field of the left inferior rectus muscle with normal and balanced rotations in the fields of the left superior rectus muscle and the right inferior oblique muscle. This permits one to feel that the right superior oblique muscle is paretic rather than the left superior rectus muscle being in essential underaction. The presence of torticollis in such a case would be of great assistance in confirming a diagnosis as to which muscle is involved. Also,

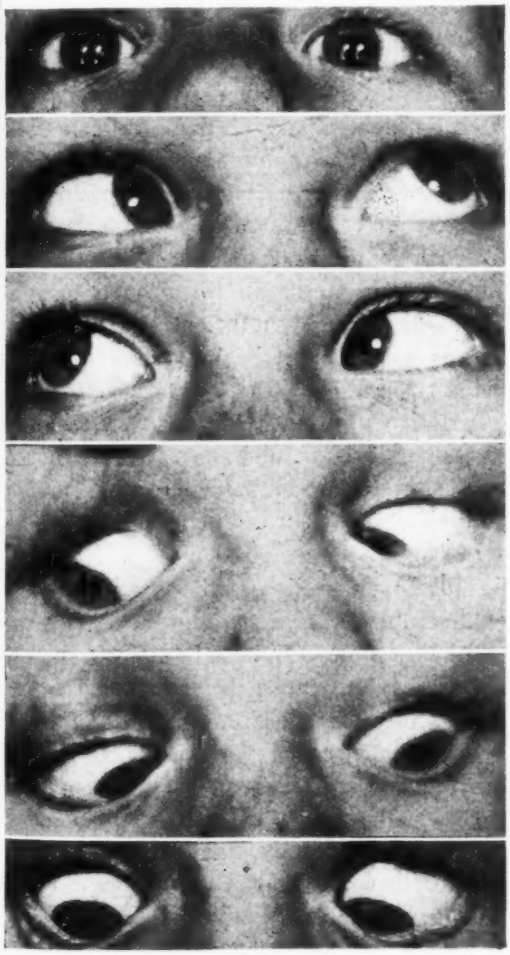

Fig. 13 (Spaeth). Bilateral limitation of equal degree in the field of the inferior oblique muscles.

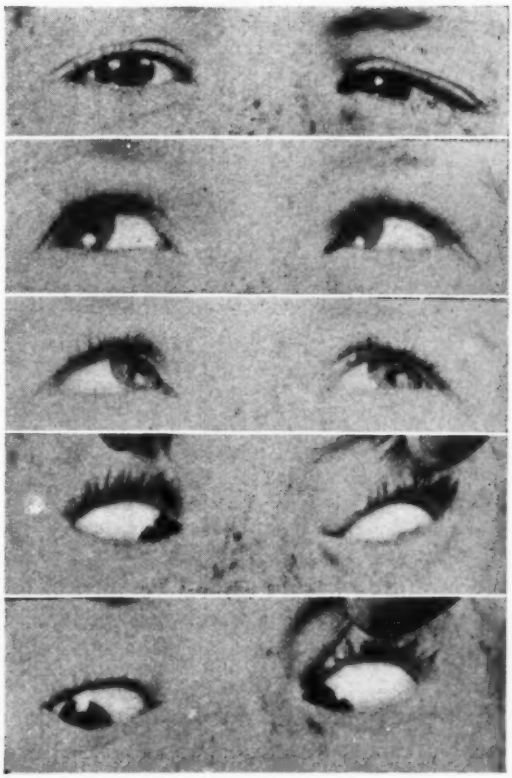

Fig. 14 (Spaeth). A monocular convergent strabismus on the left with vertical movements to the right practically equal and balanced.

if suppression and amblyopia are not present, a compensatory movement of the eye can be seen (as first noted by Bielschowsky) when the observer straightens that torticollis manually. The problem, of course, is the differential diagnosis between a paresis of a superior rectus muscle and of a superior oblique muscle. These cases (fig. 14) do not have diplopia because of suppression and, hence, amblyopia results. If that were not so, differential diagnosis would be a simpler problem. An acquired vertical muscle paralysis, by contrast, without suppression and without concomitant strabismus offers relatively few difficulties in differential diagnosis.

Figure 15 is a somewhat similar problem except here one must differentiate between a paresis of the right superior rectus muscle, the right inferior oblique muscle, and the

Fig. 15 (Spaeth). In this case it was necessary to differentiate between a paresis of the right superior rectus muscle, the right inferior oblique muscle, and the left inferior rectus muscle.

left inferior rectus muscle. The vertical deviation in the left eye could be secondary to a right inferior oblique muscle palsy. The frank limitation of movement, however, in the field of the left inferior rectus muscle with possibility of a direct antagonistic overaction of the left superior rectus muscle makes the diagnosis rather conclusive.

The differentiation of a primary and a secondary action is rather important, because surgery when necessary for a vertical

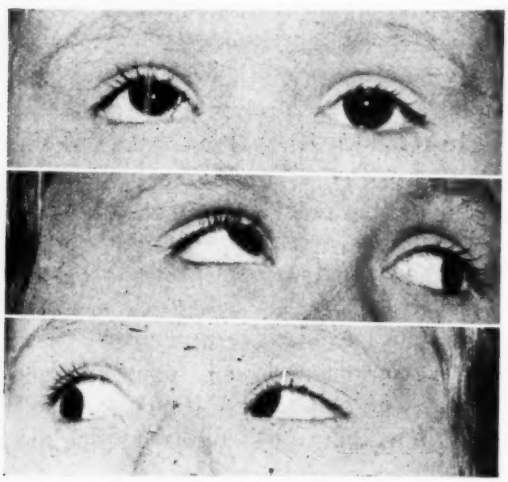

Fig. 16 (Spaeth). A frank hyperactivity (bilateral) in the contralateral rotations of the inferior oblique muscles without any lateral deviation present at any time.

component is at times successful only when applied to the conjugate overacting muscle (secondary deviation) and not to the primary underacting paretic muscle; at other times the reverse is true.

Vagaries and inconsistencies in the vertically acting muscles are not too uncommon. Figure 16 shows frank, bilateral inferior oblique muscle hyperactivity in the contralateral rotations without any lateral deviation present at any time. The refractive error was equal in both eyes. The patient may have had bilateral minimal paralyses of the two superior recti but, if so, these were equal in amount, for the measurement of the maximum contralateral hyperphoria was exactly the same in prism diopters and diplopia could not be elicited at any time. Even more significant was this, the vertical muscle situation did not result in any lateral strabismus nor in the development of subjective ocular symptoms.

Another instance follows. A patient was seen very recently with an ordinary, apparently congenital, sixth-nerve palsy on the left and without limitation of upward gaze in the midline (fig. 17). When left lateral rotations were demanded, not upward but on the horizontal level, the overaction of the internal rectus muscle on the right, consequent to the paralysis of the external rectus muscle on the left, was accompanied by an overaction of the right inferior oblique muscle (bottom view, fig. 17).

This overaction of the right inferior oblique muscle increased with attempted upward gaze to the left, even though the paralysis of the left external rectus muscle prevented the left eye from rotating into the field of the greatest vertical action for the conjugate yoke muscle of the right inferior oblique; that is, the left superior rectus muscle. One may say, therefore, that in this case a true spasm was present in the inferior oblique muscle. Normally this muscle should not be affected, in this overaction, by left lateral rotations on the horizontal level. It

seems as if the overaction of the inferior oblique muscle must be connected with the simultaneous overaction of the right internal rectus muscle. This has been suggested before in considering overconvergence.

It must be remembered, in these cases, that the patient should develop the horizontal strabismus in the eye with the greater refractive error, accompanied by sensory inhibition and amblyopia, but the vertical element connected with the case, and responsible, in part, for the lateral squint may be in either of the two eyes. This disturbance in the vertical may be found, although not as frequently, in the nonstrabismic eye as in that with the manifest squint.

Clinical experience seems to suggest that when the involved vertically acting muscle is on the eye which has the higher refractive error, then, also, one will find that the vertical component present in that case will be greater than the lateral component.

Figure 18 is a case with accidental alternating strabismus, the greater deviation being in the left eye. The vision in the right eye was 6/7.5, and in the left, 6/60, with correction. The refractive error, plus sphere and cylinder combined, was greater in the left eye. The horizontal deviation was largely present in the left eye. Nevertheless, the vertical muscle pareses were in the right superior rectus muscle and the right inferior oblique muscles. The greatest overaction present, and as a secondary deviation, was in the left inferior oblique muscle.

Figure 19 is one of a child with a paresis of the right inferior oblique muscle. The lateral deviation is seen in the right eye and it is not marked. The vertical deviation is the greater of the two. The right eye has developed amblyopia in this child and the patient consistently fixes with the left eye. The light reflexes, corneal, of the three upper photographs of Figure 19 are rather conclusive. In this case of lateral concomitant

Fig. 17 (Spaeth). A sixth-nerve palsy with normal upward gaze in the midline.

strabismus the vertical component is appreciably greater than is the lateral. This is a logical sequence of events.

One must be sure, however, not to confuse the fixing eye in such instances with a possible secondary manifest deviation, such a

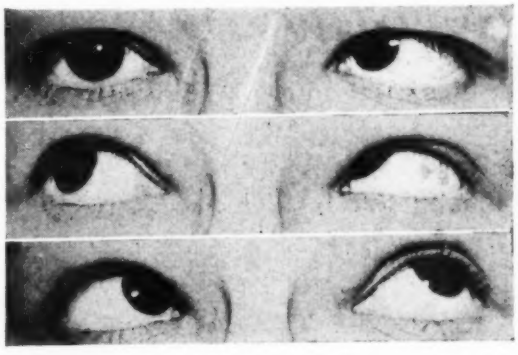

Fig. 18 (Spaeth). An accidental alternating strabismus.

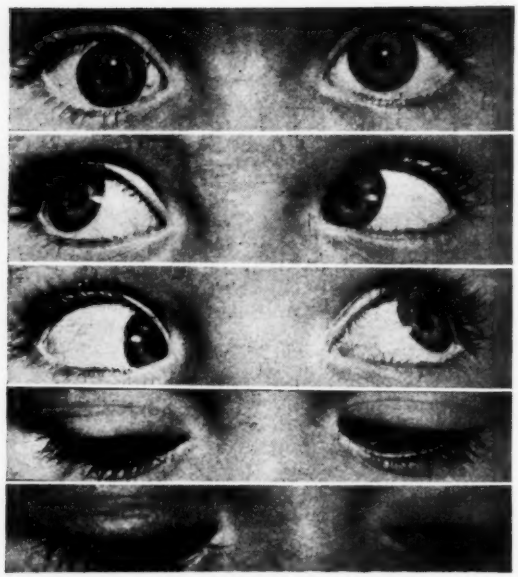

Fig. 19 (Spaeth). A paresis of the right inferior oblique muscle.

deviation being the result of fixation with the eye having the vertically paretic muscle. Figure 20 is another interesting deviation from that which one might expect. The child had a paresis of the superior rectus muscle on the right and some levator involvement as well, with a divergent strabismus on the right. There was no appreciable left inferior oblique muscle overaction, neither was there any right-sided superior oblique overaction in downward gaze. There was, however, in

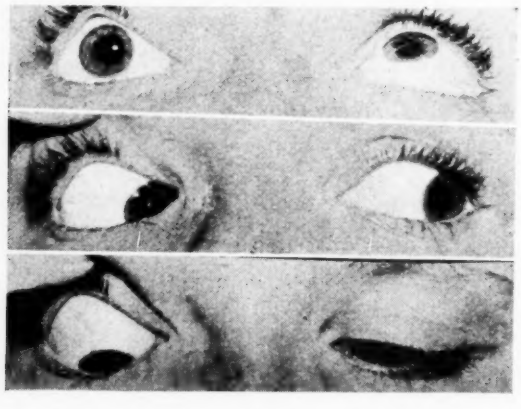

Fig. 20 (Spaeth). A paresis of the superior rectus muscle and some levator involvement, with a divergent strabismus.

the rotations directly downward, a marked overaction of the right inferior rectus muscle. In this case if a levator had not been simultaneously involved on the right, one would be suspicious of an inferior rectus fibrosis limiting the upward excursions of the right eyeball in a manner similar to a

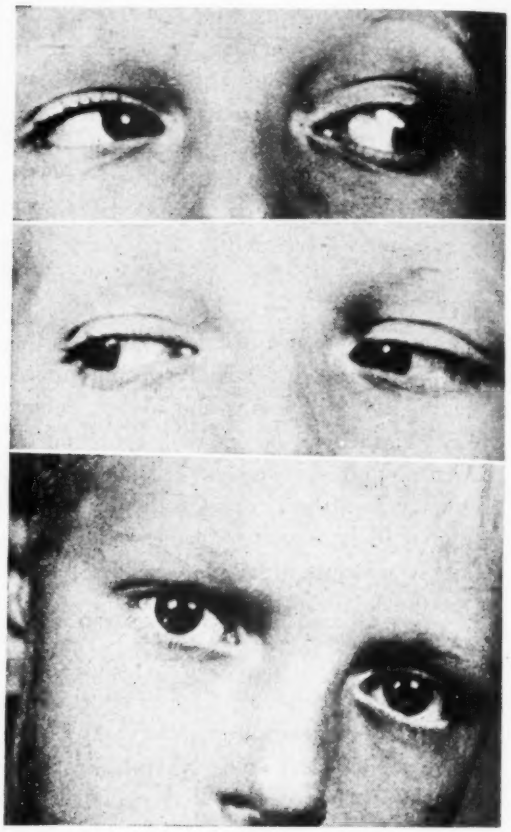

Fig. 21 (Spaeth). A monocular convergent strabismus with torticollis toward the left.

lateral retraction syndrome. Surgical examination of the inferior rectus muscle revealed a perfectly normal muscle.

These cases, if carefully observed, will frequently show torticollis to varying degrees—sometimes moderate in amount, at other times quite marked. The presence of torticollis suggests a fair prognosis for the reëstablishment of postoperative single binocular vision because it is a defense mechanism for escaping diplopia.

Figure 21 is a monocular convergent squint with a rather marked torticollis toward the left, with underaction of the right superior oblique muscle, and with normal vertical movements to the right. It was this head tipping which occasioned the patient's first visit to the oculist. (He was referred by an orthopedist.)

The various essential factors connected with and related to these cases can be uncovered only by accurate prism measurements. These should be done with screening and with prisms in the six cardinal positions, before and after cycloplegia, checked for certainty, and carefully noted upon a spider graph (that formerly suggested by Duane or the more recent one of White's are equally satisfactory). Amblyopia and suppression must be broken down so that measurements are obtained with an approximation to macular fixation, for extramacular fixation tends to exaggerate conjugate yoke muscle secondary deviations, as it changes the prism measurements of the objective and subjective angles of squint.

Recapitulation

The entire problem of a vertical component in so-called concomitant strabismus must be considered from the following standpoints.

1. *Congenital disturbances.* The likelihood that congenital disturbances in the levators, or the depressors, frequently play a profound role in the development of convergent and divergent lateral strabismus of all types, probably excepting true or essential alternating strabismus. It seems that the absence of this is an attribute of a correct diagnosis of that type of squint.

2. *The uncovering of a vertical element* in a case under consideration and the diagnosis of the primary and the secondary deviations are basic. Not only is it necessary to make a diagnosis of the type of muscle condition present but also to identify the muscle or the muscles involved. It is well to remember that when a left superior oblique muscle palsy is confused with a possible right superior rectus muscle palsy, or a left inferior oblique muscle palsy with a possible right inferior rectus muscle palsy, that the torticollis is always toward the side opposite from the eye involved. Supression of vision and amblyopia are the patient's means of fighting the cause of the torticollis, hence torticollis is not as constant a sign as it might be.

3. *Accommodative strabismus* can be complicated by an inferior oblique muscle hyperaction without demonstrable paresis of the contralateral synergist.

4. *Oblique muscle overaction* is not unusual, inferior or superior, and is due either to a paresis of the contralateral synergist yoke muscle, to a paresis of the homolateral direct antagonist, or to some mechanical defect in the orbit itself—probably connected with fibrotic bands in and to the check ligaments.

5. *A similar overaction* occurs in the inferior and superior rectus muscles as in the oblique muscles, consequent to similar factors. These cases are perhaps less common.

6. *The greatest vertical deviation* need not be present in the amblyopic eye. The horizontal squint, however, is present in that eye and this is the primary lateral deviation.

7. *Lateral concomitant deviation.* It is not always possible to state that the lateral concomitant deviation is due: (a) to the paresis of a vertically acting muscle; or (b) to the secondary deviation of a synergist to a paretic muscle; or (c) to the overaction of the direct antagonist to a paretic muscle. All three may be of significance together or at different times. Because of that the next paragraphs are perhaps the most significant in surgical therapy.

8. *The surgical therapy* for these situations is important. The results of clinical experience (and as such it should be seriously considered) has resulted in the following conclusions.

a. It is wasted time and an unnecessary expenditure of money to attempt the cor-

rection of these cases by orthoptics for they are certainly pure surgical conditions. The surgical correction should be done, if possible, before the age of four years.

b. If the vertical deviation is greater in amount than the lateral (horizontal) deviation it should be handled surgically first.

c. If the lateral deviation is the greater in degree, then that should be operated as the initial procedure. Lateral deviations tend to diminish in degree following corrective surgery to a vertical component, but vertical deviations may decrease or increase in any amount following the surgery to the lateral muscles, when that should be the primary operative procedure.

d. As the vertical component approaches the lateral component in degree or amount of deviation, then also will it be necessary to use surgery for the vertical element, either as a primary, a secondary, a simultaneous, or a subsequent operative procedure.

e. Satisfactory single binocular vision with good working fusion can never be obtained as long as a vertical component is present to an appreciable degree.

f. Minimal residual amounts of a vertical deviation lend themselves well to surgery. Surgery is only successful when applied to the proper deviation, either the primary or the secondary deviation, as indicated in any given case. Three surgical procedures are available, and at times necessary. Either the paretic muscle must be augmented in its effect, or the overacting conjugate yoke muscle limited in its overaction, or the direct antagonist to the paretic muscle in the same eye limited in its relative overaction. The following are good working rules.

Habitual fixation with the paretic eye needs surgery to the conjugate yoke muscle.

Marked torsional defects in the image, as manifested by torticollis, extensive paralysis of a muscle, and secondary overaction of the direct antagonist need surgery to the paretic muscle. This is also so when the eye without the vertical palsy is the fixing eye.

Surgery to the conjugate yoke muscle is indicated in spasm of the conjugate yoke muscle, in the lesser degrees of paralysis, and as an additional procedure to augment the effect of surgery to the paretic muscle whenever that becomes necessary because of residuals.

Surgery to the direct antagonist of a paretic muscle is indicated when spasm is present in that muscle, and as an adjunct procedure with an after surgery to the paretic muscle.

An extensive recession of a superior rectus muscle is seldom if ever indicated, and a tenotomy of that muscle is never permissible.

9. In general, *the sequence of surgical procedures for the vertical muscles* seems to give best results when applied as follows: First, (a) a recession of the overacting synergist, or (b) a resection or advancement of the paretic muscle. Second, a reversal of the sequence of a, and of b. Third, recession of an overacting direct antagonist. This is especially significant in considering the obliques. For example, the recession of an inferior oblique muscle, when overacting, will give a rather poor postoperative result, if, in the case under treatment, the proper procedure should have been the resection of the superior oblique muscle, directly antagonistic to the inferior oblique muscle being discussed. It is important that one select the proper oblique muscle for surgery in any case under consideration.

Conclusions

This presentation with the cases shown has but one purpose—to emphasize a relationship which exists between so-called concomitant strabismus and disturbances in the action of the vertically functioning extraocular muscles. This suggests a factor in the etiology of squint and, by a reversal in the sequence of events, a means for the better correction of the condition. The cosmetic appearance is not the best result obtainable in a case. Single binocular vision, or as close an approach to this as is possible, is the end result desired.

1930 Chestnut Street (3).

HISTOLOGY OF THE RETINA IN A CASE OF TAY-SACHS'S DISEASE*

JOHN A. MACMILLAN, M.D.
Montreal, Quebec

Comparatively few eyes have been examined histologically in cases of Tay-Sachs's disease, or the infantile type of amaurotic family idiocy. Werncke[1] states that not over 15 were examined up to 1939. Since then, Hagedoorn[2] and Hassin[3] have each reported the retinal findings in one case of the disease. To these I should like to add another.

Although the clinical features of the disease are well known there is still uncertainty regarding the histologic changes. Coats,[4] in particular, found only changes in the ganglion-cell layer of the retina and stated that the histologic changes found in the other layers of the infantile type were of a postmortem nature. While the delay in fixing the globe after death was in some of the reported cases so great that postmortem changes influenced the findings to a degree that conclusions could not be drawn, in others, especially in Hancock's and Coats's[5] case, where the eyes were examined immediately after death, and in Verhoeff's[6] case, where the eyes were removed 15 minutes after death, postmortem changes could not have occurred.

In the case here reported, five hours elapsed before the eyes were removed and placed in fixing fluid but the body was kept in refrigeration from immediately after death. Further, the changes were so definite that we feel they were certainly a part of the pathologic condition present and therefore add more evidence to prove that the changes are not entirely limited to the ganglion-cell and fiber layer but also that there are changes in the inner nuclear layer of the retina.

It is for this reason, and the fact that these changes lend themselves so well to reproduction, that we thought it of value to report our findings. Moreover, confusion has arisen from the use of the term "ganglion cell," which in this paper refers only to the ganglion cell of the ganglion-cell layer. While the bipolar cells of the internal nuclear layer are ganglion cells they are not usually referred to as such.

Hancock and Coats[5] state that the changes in amaurotic family idiocy are absolutely confined to the ganglion cells and their corresponding nerve fibers, yet later on they say that the ganglion cells which normally occur in the inner nuclear layer are also degenerate. This is ambiguous, but it would appear that they meant by this the Dogiel ganglion cell, or those found normally at times in the inner layer of the internal nuclear layer. The changes in the internal nuclear layer (fig. 1B) appear through its whole depth and therefore are not limited to the Dogiel cells.

The name "Tay-Sachs" is derived from Warren Tay,[7] who, in 1881, published a report of a case of symmetrical changes in the region of the yellow spot in each eye of an infant, and from Bernard Sachs,[8] who, in 1887, gave a clinical pathologic account of a similar case under the heading of "Arrested Cerebral Development." The familial characteristics of the disease were established in 1896, when Sachs[9] reported on a sister of his original patient and suggested the name "amaurotic family idiocy." Tay,[10] however, in 1884, had also reported on a third instance in the same family of symmetrical changes in the yellow spot of each eye in an infant.

The disease is defined by Duke-Elder[11] as a familial disease of early life due to

* From the Department of Ophthalmology, Royal Victoria Hospital. Presented before the III Pan-American Congress of Ophthalmology, Havana, Cuba, January, 1948.

primary lipoid degeneration of the ganglion cells of the entire central nervous system, in which the ganglion cells of the retina participate, characterized by unique bilateral fundus appearances and invariably terminating in blindness, paralysis, idiocy, and death.

These are several forms of disease due to widespread lipoid degeneration—Tay-Sachs's disease of the infantile type of amaurotic family idiocy; a late infantile type described by Bielschowsky;[12] the juvenile type also known as Batten-Mayou disease in England, Spielmeyer-Vogt, or Spielmeyer-Stock, disease in Germany; while, in America, Oatman called it familial maculocerebral degeneration.

Other diseases due to lipoid degeneration are Gauthier's disease and Neimman-Pick's disease. The last named may involve the retina as well as practically all the structures throughout the body. In an effort to simplify the various lipoid diseases in which retinal changes may be found, I have constructed Table 1.

TABLE 1

LIPOID DEGENERATION OF THE GANGLION CELLS OF THE RETINA FOUND IN AMAUROTIC FAMILY IDIOCY*

Infantile Type	Late Infantile Type	Juvenile Type	Neimman-Pick Disease
Tay-Sachs About 6 months	Bielschowsky 3–5 years	Batten-Mayou Spielmeyer-Vogt Spielmeyer-Stock Oatman Familial maculocerebral degeneration	Or Lipoid splenohepatomegaly Or Lipoid histiocytosis
Cherry red spot Jewish Female 3-1	Pigment change Nonracial	Second dentition Pigment change Nonracial	

* Also congenital type (Norman and Wood, 1941); and adult type (Kufs, 1929).

CASE REPORT

The case here described is that of a Jewish boy, who died at the age of 17 months, with all the typical signs and symptoms of Tay-Sachs's disease, including the gray area in the posterior pole and a cherry-red spot in the macula of each eye. The eyes were removed five hours after death, hardened in formalin, embedded in celloidin, and sectioned. The following stains were employed: Hematoxylin-eosin, Van Gieson, Bielschowsky, silver carbonate for oligodendroglia and microglia, gold chloride sublimate, phosphotungstic acid, hematoxylin, Nissl, Loyez, Bodian, and osmic acid.

The microscopic findings show a great reduction in the number of ganglion cells in the ganglion-cell layer of the retina (fig. 1). Most of those remaining, especially in the posterior pole, are greatly swollen and the cell bodies show many small vacuoles surrounded by slightly granular cytoplasm (fig. 2).

The cells have lost their multipolar form due to the swelling and are round in shape with the periphery of the cell clearly demarcated. In some the cell bodies cannot be made out and only nuclei can be seen but it could not be determined whether these are nuclei of the nerve cells or of neuroglia.

The nuclei of the swollen ganglion cells occupy an eccentric position in the cytoplasm and stain so densely that nucleoli are not visible. These changes are most marked in the posterior pole of the retina and, as one approaches the ora serrata, the ganglion cells are reduced in number in comparison with the normal and their cytoplasmic outline tends to be stellate rather than globular. The nuclei are not as pyknotic as those in the

posterior pole but Nissl bodies cannot be identified in the cytoplasm.

A finely granular reticulum can be made out about small vacuoles in the cytoplasm as in the nerve cells of the macular region. The nerve-fiber layer is markedly reduced in thickness but, nevertheless, a few nerve fibers are shown by the Bodian stain. There vacuolated. The vacuoles are surrounded by delicate reticular network as in the nerve cells of the ganglion cell layer.

These swollen cells are found in different levels of the internal nuclear layer (fig. 1); that is, in the inner layer where the amacrine cells, and the occasional Dogiel cells, are normally located. They are also found in the

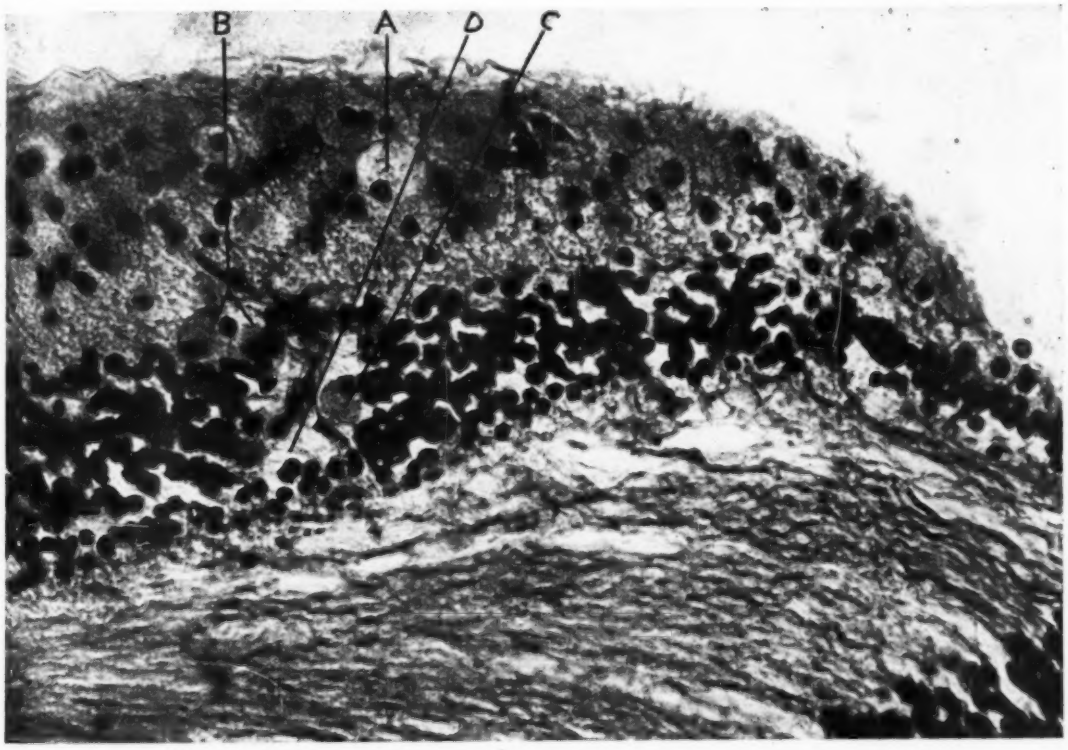

Fig. 1 (MacMillan). MARGIN OF MACULA showing inner layers of the retina. (A) Swollen ganglion cell. (B) Swollen cells in inner part of internal nuclear layer. (C) Swollen cell in middle part of internal nuclear layer. (D) Swollen cell in external part of internal nuclear layer.

is an increase in the number of nuclei in this layer which are undoubtedly glial.

There is nothing abnormal in the internal plexiform layer of the retina. The internal nuclear layer appears of about normal thickness and the cells are so packed together that only nuclear details can be made out. There are in places, however, swollen cells which resemble the swollen ganglion cell above described (fig. 3). These cells also have eccentric nuclei and their cytoplasm is middle portion which is occupied by the bipolar cell and again in the external part of this layer, the normal position of the horizontal cells.

While the bipolar cells are true ganglion cells, the term ganglion cells here is used only for those cells which are in the ganglion-cell layer.

As one approaches the periphery of the retina, the globular cells cannot be identified in this layer. The other layers of the retina;

that is, the pigment epithelium, rods, cones with their nuclei, and the external plexiform layer, are quite normal.

The nuclei in the optic nervehead are greatly increased in number and in sections the nervehead is raised above the retinal level. Nevertheless, the nerve bundles are small in diameter. In the optic nerve the nerve bundles are small, the number of nuclei in the septa is increased, the septa are thickened, and there are clear spaces between the nerve bundles.

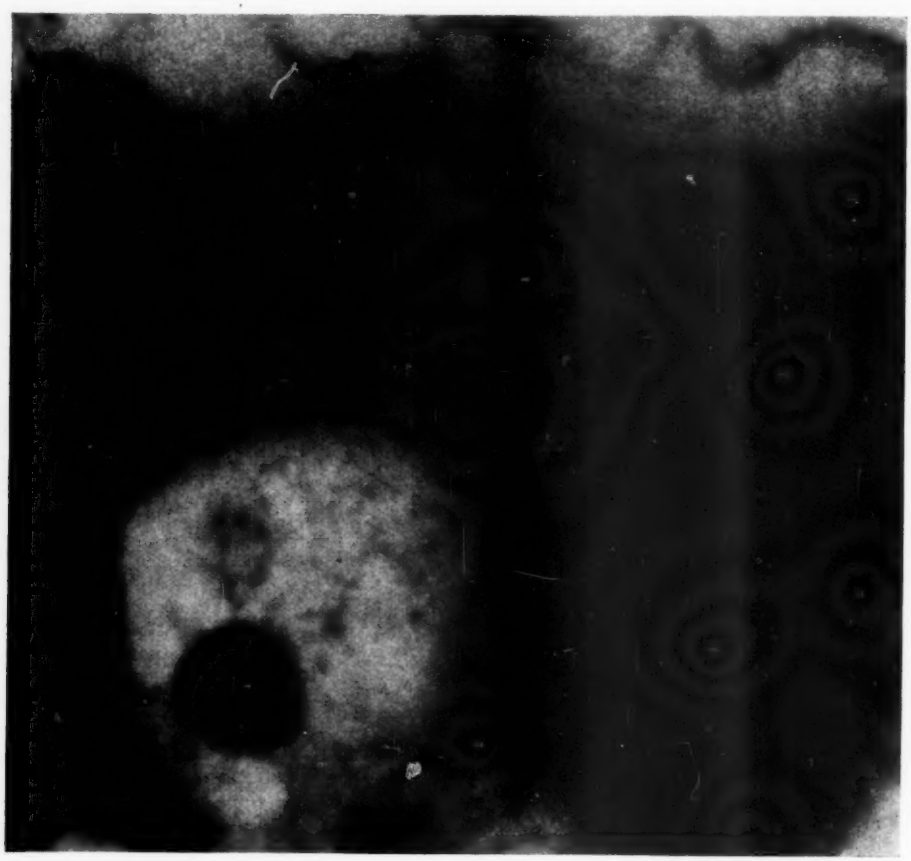

Fig. 2 (MacMillan). Higher magnification of (A) in Figure 1.

Comments

The changes in the ganglion cells of the ganglion cell layer and of the nerve cells of the internal nuclear layer are similar, although they are fewer in the internal nuclear layer and they are found only in the posterior pole of the eye. The question can be raised as to whether they are nerve cells. Their location in these two areas is presumptive evidence in a patient with amaurotic family idiocy. They cannot be cells of the reticuloendothelial system or microglia as morphologically they differ greatly. The vacuoles are numerous and small with wide bands of delicately granular cytoplasm surrounding them, while the microglia in the "gitterzellen" phase show large vacuoles, delicate trabeculae and irregularly shaped pyknotic nuclei. The nerve cells of the cerebral cortex and spinal cord show identical globose swelling of their cell bodies. However, in many of these cells in the central nervous system, nuclear detail can be made out.

Fig. 3 (MacMillan). Higher magnification of (B) in Figure 1.

The marked atrophy of the optic nerve, as seen by the small nerve bundles, the disturbed architectural pattern, the apparent connective-tissue increase, and the increased nuclei in the glial septa and the spaces between the bundles in the nerve, is the typical picture of optic-nerve atrophy which is the result of atrophy of the axis cylinders from the abnormal ganglion cells.

Summary

For years it has been generally accepted that lipoid deposits occur in the ganglion cells in amaurotic family idiocy. There are many types of ganglion cells in the central nervous system and in the retina. Among these the bipolar cells of the retina must be classed as neurones or ganglion cells. The globular cells of the internal nuclear layer must be changed bipolar cells because of their location. It is admitted that some may be changed horizontal neurones and others changed displaced ganglion cells or so-called Dogiel cells. The point is that these swollen cells are found in all depths of the internal nuclear layer—inner, middle, and outer layers. It is the changes in this layer which, although described before, because of their clarity and readiness of illustration justify this report. It is not surprising that changes should occur in this layer as the cells are neurones in the same sense as the ganglion cells in the ganglion cell layer.

Cytologic studies of the retina are difficult; we have adhered to methods which do not lend themselves to detailed cytologic

study. New methods of fixation of the retina and improved technical methods must be devised. In this particular case we regret that more detail could not be given of the cell changes. Many of the stains carried out were disappointing because the proper fixative necessary for different stains had not been used.

We should not stop at histologic description of the lesions in amaurotic family idiocy, important though they be, but we should look for metabolic abnormalities in the disease. We ordinarily think that ganglion-cell activity pertains only to nerve impulses and often forget that ganglion cells, like other cells, must carry on cell metabolism. Many neuro-histologists feel that myelin is formed by the neurones when their axis cylinder processes are to be insulated. If this be so, myelin, a complicated lipoid, is synthesized by metabolic processes going on in the nerve cell. Theoretically then, all nerve cells, even though their axones be unmye'inated, may be suspected of some such metabolic activity. It would seem that the abnormal material in the ganglion cells must be the result of an inherited metabolic dyscrasia.

Royal Victoria Hospital.

I am deeply indebted for the coöperation supplied by Dr. William V. Cone, whose knowledge of the cytology of the central nervous system has made this presentation possible.

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THE EFFECT OF EXPOSURE TO SUNLIGHT ON NIGHT VISION*

SELIG HECHT,† PH.D., C. D. HENDLEY,‡ PH.D., SHERMAN ROSS,§ PH.D. AND P. N. RICHMOND‖

I. NATURE OF PROBLEM

Reports from North Africa and the South Pacific during the last war suggested that night vision deteriorates after exposure to very strong sunlight, particularly on coral islands. Derman[2] (also reference 7) reported 50 cases of night blindness in the Turkish army, apparently due to bright light. In view of the military importance of this problem, it was decided to investigate the effects of sunlight in a quantitative manner. The problem is a three-fold one. First, is the onset of dark adaptation delayed after exposure to bright sunlight? Second, is the process slowed up so that the threshold is still above normal even after an hour or two in darkness? Third, is the effect of daily exposure cumulative?

The first question has already been answered. The speed of dark adaptation is strongly influenced by the brightness of the preceding light adaptation.[3, 4, 10] Thus, after light adaptation to 100 millilamberts (ml.), the rod portion of dark adaptation appears almost at once, whereas after exposure to 40,000 ml. it does not become evident for 12 minutes.

Previous work[4] also showed that after adaptation to high intensities the final threshold may be delayed out of all proportion to the delay in onset of dark adaptation. For instance, 40 minutes after exposure to 40,000 ml. the eye is still about 0.5 log unit above its normal final threshold. No quantitative data on the cumulative effect of sunlight were known. Our aim, therefore, was to find out how long the threshold remains above normal after exposure to sunlight, and whether such effects accumulate when people are exposed day after day.

II. APPARATUS AND METHODS

The measurements were made with two Hecht-Schlaer adaptometers, model 3. The binocular threshold of a retinal area 7° above the fovea and 3° in diameter was measured. One instrument used blue light of dominant λ 480 mμ., the other used white light. Since the intensities are given in micro-microlamberts in terms of photopic brightness, the thresholds on the two instruments will differ because of the Purkinje phenomenon. Many comparisons showed the average threshold to be 1.20 log unit higher on the white instrument, with little individual variation. All measurements were referred to the blue instrument. Each subject was measured consistently on the same instrument.

In determining a threshold the operator controls the brightness of the stimulus with a neutral wedge, while the subject operates a shutter that gives reproducible 0.2 second flashes. The wedge is first set to give a brightness about 1 log unit above threshold, and the brightness is then reduced in steps of 0.5 to 0.2 log unit, until he fails to see even a faint flash of light. With the threshold thus roughly determined, the operator varies the

* From the Laboratory of Biophysics, Columbia University, New York City, and the Medical Field Research Laboratory, Camp Lejeune, North Carolina. Part of this work was done under a contract, recommended by the Committee on Medical Research, between the Office of Scientific Research and Development and Columbia University. The measurements were made at the Medical Field Research Laboratory, Camp Lejeune. Opinions expressed here are those of the authors and are not official with the Navy Department. This work has been reported to the Committee on Medical Research on March 24, 1945.

† Due to the untimely death of Professor Hecht on September 18, 1947, this report has been prepared by his collaborators.

‡ Now at the Department of Physiology, The Ohio State University, Columbus, Ohio.

§ Lieutenant (j.g.) H(S) USNR; now at Bucknell University, Lewisburg, Pennsylvania.

‖ Pharmacist's Mate 2/c, USN-I; now at 15 School Street, Boston, Massachusetts.

brightness in a random way above and below the threshold, always by even tenths of a log unit. The point at which about 3 out of 5 exposures are detected as flashes of light is the threshold. After a minute's rest the procedure is repeated. Almost always, the thresholds agree to 0.1 log unit. When the difference was greater, more determinations were made after a longer rest, until a satisfactory threshold was obtained.

III. The course of dark adaptation following bright outdoor light

Experiments were conducted in New York on the effect of exposures to bright sky on dark adaptation. The subjects looked at the sky for periods of from four minutes to an hour. The brightness varied from 3,000 to 12,000 ml., the limiting factor being the subject's ability to look at the light without undue pain. In the final stage of light adaptation, one eye was closed to reduce the discomfort and to make it possible to look at a brighter part of the sky. Both rod and cone adaptation were followed, using the Hecht-Shlaer model 1 adaptometer,[6] with monocular observation.

At Camp Lejeune, a further study of comparatively short exposures to bright light was made, the sky brightnesses ranging from 3,500 to 16,000 ml., and the time of exposure from 2 to 35 minutes. For comparison, dark-adaptation curves were obtained after exposure to 5 and to 50 ml. In each case, the threshold after one hour of dark adaptation was obtained before the start of the light-adaptation experiment. If the subjects had to be outdoors before this initial measurement, they wore red goggles to prevent uncontrolled exposure to bright light.

TABLE 1

The delay in dark adaptation following exposure to bright sky

Subject	Time of Preliminary Light Adaptation	Time of Final Light Adaptation	Brightness of Sky During Final Light Adaptation millilamberts	Rise in Rod Threshold ½ hr. in the dark	1 hr. in the dark	2 hrs. or more in the dark
A.E.	3 hours	4 minutes	3,000 to 10,000	0.28	0.10	0.12
M.Y.	3 hours	4 minutes	3,000 to 10,000	0.45	0.09	0.04
B.L.	6 hours	none	none	0.09	0.00	0.00
B.L.	4 hours	4 minutes	3,000 to 10,000	0.55	0.16	0.16
D.H.	23 minutes	9 minutes	1,000 to 4,000	0.27	0.08	——
C.H.	2½ hours	5 minutes	10,000	0.20	0.00	0.00
C.H.	30 minutes	5 minutes	10,000 to 12,000	0.43	0.38	0.32
C.H.	20 minutes	10 minutes	10,000 and over	0.61	0.23	0.34
Y.H.	25 minutes	5 minutes	3,000 to 10,000	0.19	0.17	0.17
Y.H.	25 minutes	5 minutes	5,000	0.40	0.33	0.11
Y.H.	25 minutes	7 minutes	3,000 to 6,000	0.23	−0.04	——
S.H.	none	20 minutes	not determined	1.2	0.15	0.05
W.N.	7 minutes	4 minutes	6,500	0.55	0.25	——
W.N.	10 minutes	8 minutes	5,000	0.35	0.10	0.00
W.N.	none	30 minutes	3,500	0.70	0.34	0.20
W.N.	15 minutes	15 minutes	6,000	0.65	0.20	0.00
W.N.	10 minutes	10 minutes	9,000	0.60	0.19	0.00
W.N.	none	10 minutes	5,000	0.60	0.16	0.00
G.L.	none	30 minutes	8,000	0.30	0.30	0.02
G.L.	none	37 minutes	9,000	0.48	0.25	0.20
A.C.	none	25 minutes	16,000	1.05	0.84	0.57
Average				0.48	0.20	0.1

Table 1 summarizes the main features of the experiments. Since different subjects were used in the various tests, and there is considerable individual variation, the results for different times and brightnesses are not directly comparable. However, it is clear

enough that, after exposure to bright skylight, dark adaptation is considerably delayed. The significant parts of four representative dark-adaptation curves are shown in Figure 1.

IV. Preliminary experiments on prolonged exposure to sunlight

In these experiments each subject had his threshold measured in the morning after one hour of dark adaptation. The group then spent several hours on an ocean beach in the sunlight. After 2 to 5 hours the group returned to the dark room, and was tested after various lengths of time in the dark. Four groups were also measured the next morning after one hour of dark adaptation. Table 2 gives the results. It is clear that exposure to sunlight delays the course of dark adaptation. There seems to be some residual effect after several hours, and perhaps even overnight.

V. Final experiments at Camp Lejeune

A. Procedure

A series of systematic experiments were set up to determine whether or not daily exposures to sunlight produced any cumulative effect on night vision. We worked with 51 young men with normal night vision. The control group of 20 men was kept at indoor tasks to prevent exposure to bright light. They wore red dark-adaptation goggles whenever they went outdoors during the day. The experimental group of 31 men was sent every day except Sunday to the beach.

On sunny days near noon, the illumination was about 10,000 foot-candles, but because the sand was rather dark—having a reflectance of 0.3 at most—the brightness of the beach was less than 3,000 ml. Viewed from the beach the water was darker than the sand. The sky varied from about 500 to 15,000 ml.

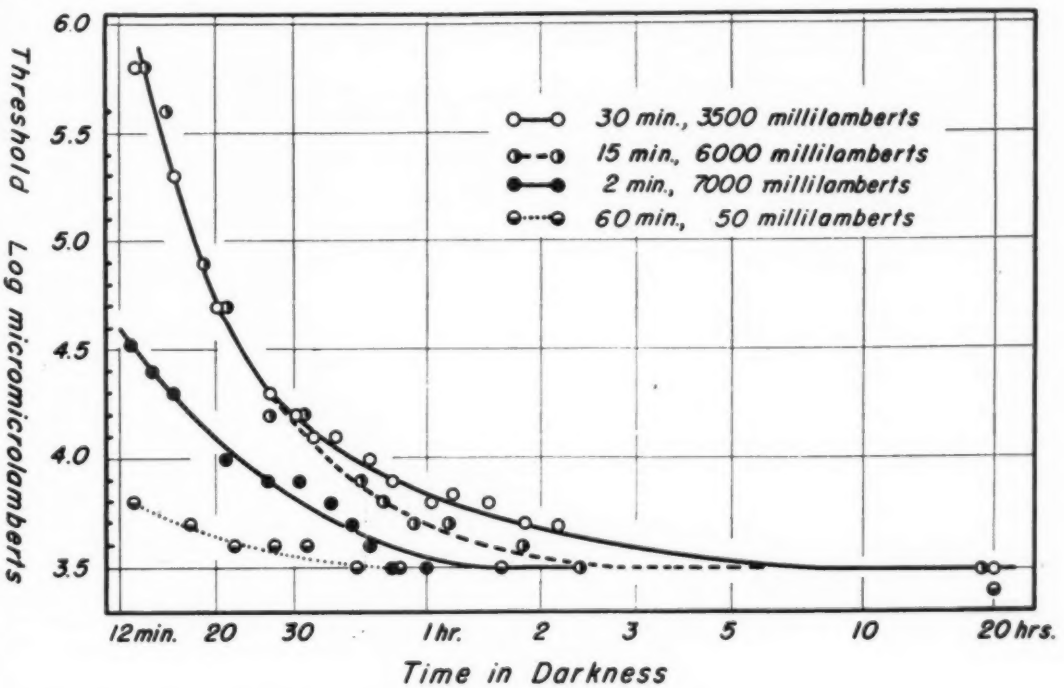


Fig. 1 (Hecht, Hendley, Ross, and Richmond). Dark adaptation after exposure to bright sky.

TABLE 2

EFFECT OF SUNLIGHT ON NIGHT VISION*

No. of Subjects	Hours Exposure	Threshold Rise—Log Units Hours after Exposure				
		½	¾–1	1½	4½–5	16+
6	2		+0.25	+0.20		−0.02
6	5	+0.85	+0.25		+0.09	
4	2		+0.27		+0.27	+0.16
6	2½		+0.23			
11	2½		+0.19			+0.09
10	5		+0.12			−0.05

* Sky Brightness 3–5×10³ ft. lamberts
Water Brightness 1–3×10³ ft. lamberts
Sand Brightness 1–2×10³ ft. lamberts

At the beach the men were instructed not to close their eyes, or protect them from light in any way. During the first 2 weeks the men were exposed to the sun for about 4 hours, of which two were spent in an open boat going to and from the beach. Later the subjects had about 6 hours of continuous exposure, of which only one was spent in transit. The subjects were dark adapted for one hour before their thresholds were measured.

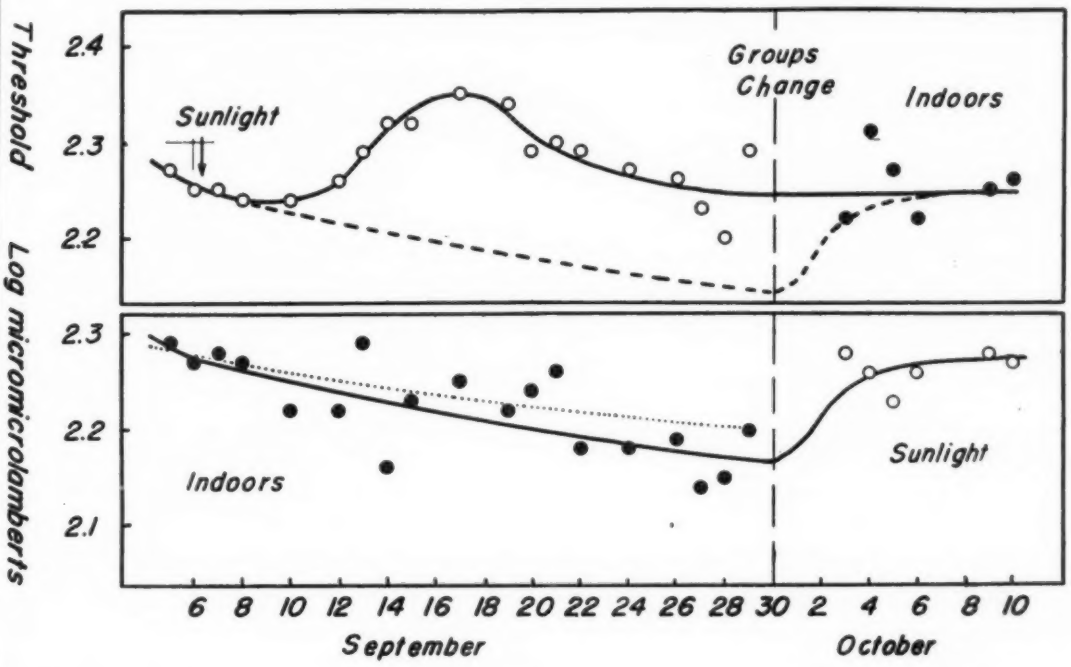
These procedures began September 5th and continued through September 30th. Af-

TABLE 3

MORNING THRESHOLDS AFTER ONE HOUR IN DARKNESS

Date		X-1	X-2	X-3	Average of X Groups	C-1	C-2	C-3	Average of C Groups
Sept.	5	2.27	2.25	2.30	2.27	2.29	2.26	2.31	2.29
	6	2.23	2.24	2.29	2.25	2.29	2.22	2.29	2.27
	7	2.30	2.20		2.25	2.26	2.29		2.28
	8		2.23	2.25	2.24		2.28	2.27	2.27
	9			2.24				2.21	
	11	2.24			2.24	2.23			2.22
	12	2.26	2.27		2.26	2.23	2.20		2.22
	13		2.32	2.25	2.29		2.28	2.31	2.29
	14	2.32		2.32	2.32	2.16		2.16	2.16
	15	2.36	2.29		2.32	2.18	2.28		2.23
	16		2.25				2.26		
	18			2.44	2.35			2.24	2.25
	19	2.22		2.47	2.34	2.18		2.25	2.22
	20	2.27	2.31		2.29	2.23	2.25		2.24
	21		2.28	2.32	2.30		2.32	2.21	2.26
	22	2.27		2.31	2.29	2.17		2.18	2.18
	23	2.25				2.14			
	25		2.28		2.27		2.23		2.18
	26		2.24	2.29	2.26		2.21	2.16	2.19
	27	2.12		2.34	2.23	2.13		2.16	2.14
	28	2.17	2.22		2.20	2.09	2.21		2.15
	29		2.27	2.32	2.29		2.28	2.12	2.20
	30			2.36				2.15	
Oct.	1	X Groups come indoors; C groups go out to beach.							
	2	2.27				2.16			
	3	2.22	2.22		2.22	2.25	2.31		2.28
	4		2.28	2.35	2.31		2.31	2.20	2.26
	5	2.14		2.40	2.27	2.22	2.28	2.19	2.23
	6	2.18	2.25		2.22	2.27	2.29	2.23	2.26
	7		2.22			2.29			
	9	2.15		2.38	2.25	2.29	2.39	2.17	2.28
	10	2.16	2.26	2.36	2.26	2.24	2.33	2.23	2.27

ter the morning measurements on September 30th, the indoor and sunlight groups changed places. The X (experimental) group, which had been outdoors every day, remained indoors. The C (control) group, on the other hand, took off their goggles and went outdoors on the beach. This continued until October 10th when the experiment had to be ended.



Sunlight Group ..... Control group curve adjusted to level of sunlight group at start
Indoor Group - - - - Randolph Field Cadets

Fig. 2 (Hecht, Hendley, Ross, and Richmond). Effect of sunlight on morning threshold.

B. Cumulative effects

Table 3 shows the morning one-hour thresholds after a night's sleep and before any exposure to sunlight. There are several ways in which the measurements may be treated, from following each small group to making weekly averages of all groups. We find the results are much the same any way the data are examined. The data are summarized graphically in Figure 2.

Initially the two groups were chosen to have almost identical average thresholds. Actually they differed by only 0.02 log unit. During the course of the experiment, the control group threshold decreases gradually; while the sunlight group threshold rises and then seems to return about to its original level. The decrease in the control group represents the improvement which comes with practice in untrained individuals. It occurs regularly, and is of the same order of magnitude as found here. For comparison, the data for 37 aviation cadets at Randolph Field are shown in Figure 2.

When the two groups were reversed the old control-group threshold (now the sunlight group) began to rise. After three days the rise was almost 0.1 log unit and further exposure caused no significant further rise. The original sunlight group (now indoors) showed practically no change for the 10 days they were protected from light.

In making a quantitative estimate of the changes caused by sunlight, we must include

the improvement shown by the control group. There is no reason to suppose that the sunlight group did not improve in a similar way. Indeed, this group did show a slight decrease in threshold the first few days. Thus the control data provide a base line for the sunlight data. This is indicated by the dotted line under the sunlight measurements in Figure 2.

Another way to show the cumulative effect of exposure to sunlight is to compare the distribution of thresholds before the experiment with those after the two groups diverged. In Figure 3 those comparisons are made, and as before the sunlight group shows a small rise in threshold after 9 days outdoors, even though 4 of these days were overcast. The outdoor group was lower than the control by 0.02 log unit at the start, but is 0.12 log unit higher after exposure. This change is 4.3 times the standard error of the difference between the two means after exposure, and the difference is certainly significant by this test.

C. Daily effects

For 9 days, between September 25th and October 5th, for each group that was exposed to sunshine, we measured the threshold after one hour of dark adaptation in the morning before the exposure, in the afternoon after returning from the beach, after supper, and early the next morning. Each indoor group was measured at the same time. The averages of these nine series of determinations are shown in Figure 4. The one-hour threshold is raised by 0.14 log unit due to sunlight, and this rise does not completely disappear until some time in the night. By the next morning the one-hour threshold is practically the same as the day before.

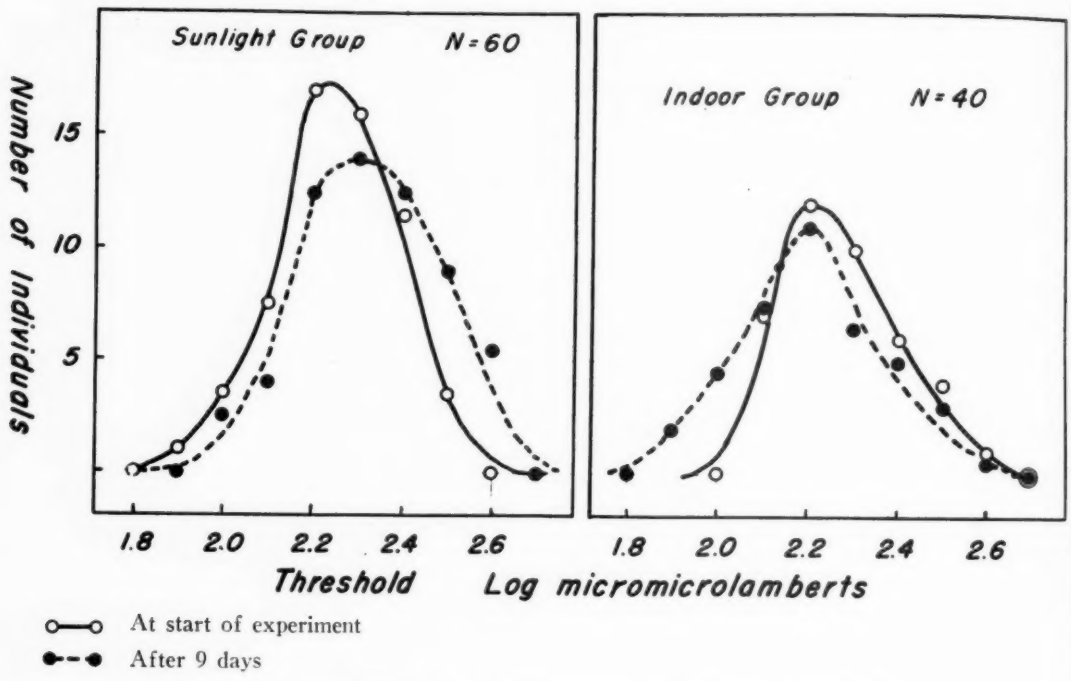


Fig. 3 (Hecht, Hendley, Ross, and Richmond). Distribution of thresholds.

VI. Discussion

From all these experiments we may conclude that night vision is interfered with significantly but not drastically for a period of several hours after a single prolonged exposure to sunlight. Our work also showed that daily exposures to sunlight produced a

cumulative chronic effect on night vision which after 10 days had about the same magnitude as the maximal temporary effect of a single exposure.

This second conclusion has been called into question by two later reports. Clark, Johnson, and Dreher,[1] working at Pensacola, Florida, confirmed the temporary effects of sunlight, and found a threshold elevation of about 0.13 log unit even after a night of dark adaptation. However, they found that "the threshold returns to normal after one day's protection from the sun." Ross, Mueller, and Richmond,[9] at Camp Lejeune, also found temporary effects that did not, however, persist overnight.

In both the Camp Lejeune studies we were handicapped by cloudy weather and by the low reflectance of the beach. The Pensacola workers did not run into these difficulties and, although no brightness measurements are reported, they state that "the bright sunlight and high reflectivity of the sand on the beaches closely approximate conditions of illumination found in the islands of the South Pacific." In view of the importance of determining for certain whether bright sunlight can cause prolonged deterioration in night vision, we feel that further investigations should be made, in tropical or arctic regions.

We may state with assurance, on the basis of the work to date, that daily exposure to bright sunlight for 3 hours or more causes a rise in threshold which is of the order of 0.2 log unit, on the average, during the succeeding night. Such a rise has a substantial influence on night visual performance. It has been shown[5] that a person so affected by sunlight has his night visual acuity cut in half, his range decreased to half, and the contrast which he can just recognize increased by 50 percent by comparison to his normal self. This deterioration in visual function is of about the same order of magnitude as that suffered by an aviator flying at night between 12,000 and 15,000 feet without oxygen.

While is it not our object in this paper to inquire into the physiologic mechanisms involved, we are inclined to believe that it is the visible part of the radiation that is effective. Ultraviolet forms only a small part of the radiation reaching the cornea, and while it can burn this part of the eye, as in snow blindness, it does not reach the retina to any appreciable extent, because of the great absorption of the lens and eye media. Nevertheless, if any further sunlight studies are made, it might be well to exclude the ultraviolet, merely to have the information. Infrared rays, although they comprise over half the radiation on a sunny day and do get through to the retina, are probably also not responsible. We know of no evidence that infrared radiation is harmful unless many times as intense as it is in the brightest daylight. There are cases reported of retinal burns from staring at the sun, but this did not occur in our experiments.

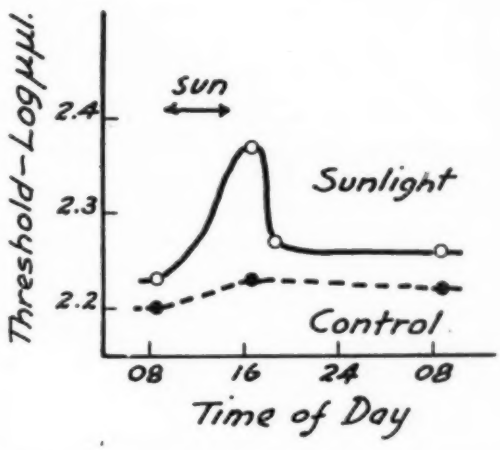


Fig. 4 (Hecht, Hendley, Ross, and Richmond). Thresholds after one hour in darkness.

There exists an extremely simple preventative for avoiding the ill effects of exposure to sunlight, namely adequate sunglasses. Many of the best known and most expensive sunglasses now available are designed for maximum transmission of visible radiation, while cutting out as much as possible of the ultraviolet and infrared. As a result, they trasmit 50 or even 75 percent

of the light. Such sunglasses would be of little value in preventing injury to night vision. It was recommended[5] that glasses should transmit at most 10 percent of the light. The Navy did issue sunglasses that came close to this specification, having a transmission of about 12 percent (Sun, N-1, Contract No. NXsX-66884).[8] Clark *et al.*[1] found that these glasses did indeed protect against the deleterious effects of sunlight on night vision.

VII. Summary

1. Exposures to ordinary sunlight produced temporary and cumulative effects on night vision. A single exposure of 2 or 3 hours delayed the onset of rod dark adaptation by 10 minutes or more, and slowed the process itself so that the normal night vision threshold was not reached for several hours afterward.

2. After repeated daily exposures to sunlight, the delay in reaching the normal threshold persisted overnight, and the threshold after complete dark adaptation rose higher each day for about 10 days and remained at a higher level.

3. This elevated threshold corresponds to an average deterioration of about 50 percent in visual acuity, range of visibility, contrast discrimination, and in the frequency of picking up a target when it is barely visible. The effect shows considerable individual variation, but the average loss in night vision is nearly the same as is suffered by flying at 12,000 feet at night without oxygen.

4. It is strongly recommended that sunglasses transmitting 10 percent or less of visible light be used by all persons who, while working in bright sunlight during the day, will be expected to perform critical night duties soon afterward.

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UVEOPIGMENTARY SENSITIZATION*

HAROLD H. JOY, M.D.
Syracuse, New York

SYMPATHETIC OPHTHALMIA

Although anaphylaxis as a cause of sympathetic ophthalmia had been discussed earlier, Elschnig[1] was the first to present a clear description of a workable theory supported by experimental facts. In 1910, he advanced the idea that sympathetic ophthalmia was the result of an anaphylactic reaction in which uveal pigment acted as the antigen. His theory assumed that a perforating wound involving the uvea may cause liberation, dissemination, and absorption of the pigment. Since the uvea is the only structure containing this pigment, he postulated that its absorption produced hypersensitivity to it of the uvea of both eyes. He maintained that further absorption acted as a shocking dose, causing an anaphylactic intoxication of the sensitized uveal tissue of the uninjured eye, and resulted in the clinical picture of sympathetic ophthalmia.

ALLERGIC THEORY

Elschnig's theory was based on complement-fixation studies indicating that the pigment of the uveal tract was the uveal constituent responsible and that it was capable of acting as an antigen in the homologous animal, and that it was organ specific but not species specific. These findings were confirmed by Weichardt and Kummel,[2] and later by Kummel,[3] but were questioned by Rados,[4] Fuchs and Meller,[5] and von Szily.[6] In 1916, Woods[7] repeated Elschnig's work, and, with one minor exception regarding species specificity, confirmed his original results. These experiments were again repeated and confirmed by Nakamura,[8] and later partially confirmed by Kodama.[9] In studies of the mode of absorption of uveal pigment in man, Henton[10] offered further proof of its organ specificity.

Since 1916, Woods has taken the dominant role in the investigation of the antigenic properties of uveal pigment, and the present status of the allergic theory is largely due to the results of his studies. The immunologic properties of uveal pigment have been substantiated, and it has been established that after an injury to the uveal tract, the pigment may be absorbed and produce an immunologic reaction. Woods[11] showed that it was possible to produce uveitis in the fellow eye after intraocular sensitization of dogs with uveal pigment and subsequent inoculation by intraperitoneal injection. He also found that antibodies specific for uveal pigment were usually present in patients whose eys healed normally, but were absent when the inflammation was prolonged and when sympathetic ophthalmia developed.[12] These findings were not entirely confirmed by Fodor[13] who, while he demonstrated the occurrence of such antibodies in many cases, did not concur as to their appearance in specific conditions. He agreed as to their absence in sympathetic ophthalmia, but he found them only irregularly in eyes that healed uneventfully, and also found them in some cases of retinitis pigmentosa and in other diseases of the uveal tract. Gifford[14] failed to find antibodies in four patients, two of whom had sympathetic ophthalmia. With the development of more modern immunologic ideas, Woods[15] later concluded that he had exaggerated the significance of the appearance of antibodies in his earlier studies.

INTRADERMAL SENSITIVITY TEST

Because of the technical difficulties and the delay in the appearance of the antibodies in the complement-fixation reaction, Woods[16] supplanted it by the less complicated and

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quicker acting intradermal sensitivity test. Using uveal pigment as an antigen, Woods and Little[17] revealed several significant features. They found that true hypersensitivity to uveal pigment occurred only after penetrating wounds of the uveal tract, and that in such cases an unfavorable prognosis could almost always be expected. The response in patients with sympathetic ophthalmia was generally positive, but occasionally a negative reaction was found during acute exacerbations. This led the authors to assume that there may be periods showing a negative phase similar to those found in certain stages of acute tuberculosis, in which the reaction to tuberculin may be negative.

"False positive" test

It has long been noted that occasionally patients with severe traumatic uveitis, who do not develop sympathetic ophthalmia, show a positive cutaneous pigment reaction, the so-called false positive test.[16] Some eyes from such patients have been removed because of the fear of sympathetic inflammation, and others because of pain due to severe uveitis. The pathologic picture in such eyes differs somewhat from that of sympathetic ophthalmia. Nevertheless, at one time consideration was given to the possibility that sympathetic uveitis might have occurred if the eyes had not been removed.[16] Later, it became apparent that this condition did not necessarily predispose to sympathetic ophthalmia, for in cases in which the affected eye was not removed, the fellow eye did not become involved. Woods[16] has long been impressed by the clinical similarity of these cases and he and Little[17] suggested that the condition appeared to be a clinical entity, the characteristics of which are the development of a delayed, severe, recurrent, and chronic postoperative or traumatic uveitis associated with the synchronous development of allergy to pigment.

Histologic studies

The evidence of relationship of uveal pigment allergy to sympathetic ophthalmia received considerable support from Jonas Friedenwald's histologic studies.[18] Since, according to the Elschnig-Woods theory, uveal pigment is the antigen responsible for the pathologic reaction in sympathetic ophthalmia, Friedenwald speculated that pigment granules within the phagocytic cells should be found in the centers of the lesions; for if the disease is allergic, the specific antigens should be demonstrable in the specific lesions.

In his investigation, he found that not only were the epithelioid cells and giant cells of the uveal tract filled with stainable granules, but so were those in episcleral and retinal nodules. The retinal findings were particularly significant, for the picture was quite different from that found in other pigmentary disturbances of this tissue. The characteristic feature was the fact that whenever melanin pigment was deposited in the retina, the granules were engulfed by epithelioid cells and giant cells and surrounded by an inflammatory infiltration which had all the characteristics of the specific lesions of sympathetic ophthalmia.

The presence of these nodules raised the question as to how far the characteristic inflammatory reaction of sympathetic ophthalmia might be considered as due solely to an allergic reaction to the pigment granules. The question was answered by studying histologically the reaction in the skin to a local injection of a suspension of uveal pigment. Friedenwald found that two weeks after injection the histologic picture of the excised skin was identical to that of the ocular reaction in patients with active sympathetic ophthalmia. On the other hand, in normal and insensitive individuals, the picture was quite different. He concluded that the disease had been reproduced in the skin of a susceptible individual by the local injection of the presumed specific antigen. He further concluded that the characteristic pathologic changes found in this disease are compatible with the allergic hypothesis. Alexseev[19] made similar histologic examinations of the excised cutaneous segments in 6 cases, 5 of

which showed clinically positive skin tests. The one clinically negative case proved to be positive histologically.

Desensitization

On the assumption that allergy to uveal pigment is at least a fundamental factor in the production of sympathetic ophthalmia, Woods[16] presumed that, theoretically, this factor should be removable by desensitization with the specific allergin. Using uveal pigment injected intramuscularly he sought to specifically desensitize patients with sympathetic inflammation. As the result of many years' observation involving a comparatively large number of patients, he believes the therapy to be specific and of true value.[17] However, uveal pigment is an insoluble and feeble antigen, and its absorption is slow so that desensitization is a long-drawnout process.[20] Friedenwald[18] obtained a more rapid desensitization by exposure of the skin of the body to ultra-violet light. His purpose was to produce a proliferation of the melanophores of the skin, so that the body might be provided with a readily available and widely distributed supply of pigment antigen.

Comment

Thus, it appears that the proponents of the allergic theory have built up a strong case in support of their contention that uveopigmentary sensitization is the essential element in the production of sympathetic ophthalmia. However, this evidence is not quite as formidable as it first appears. Its cornerstone is the assumption that uveal pigment is the antigenic substance involved in the process. But, in spite of extremely strong evidence that this is true, the fact that some investigators have been unable to confirm its immunologic reaction cannot be ignored.

Even if one accepts the organ-specific properties and antigenic action of uveal pigment, consideration must be given the possibility that the immune response is not specific, but may be due to the absorption of a foreign protein. It is possible that some substance other than the pigment is the antigenic agent. The isolation of uveal pigment is an exacting and complicated process and there is always the chance that it may not be free from other tissue substances or that changes may occur during its preparation.

Moreover, it is a well-known fact, as pointed out by Berens,[21] that allergic individuals give a reaction to certain, sometimes many, antigens, and that those who are nonallergic react negatively to the same antigens. The possibility arises that the former group may have a tendency to react positively and the latter group negatively to uveal-pigment injections.

Whatever the reasons, some investigators[14, 19, 22–25] have not found the uniform clinical results, in using the skin test, noted by the Wilmer group and by Gill.[26] It is possible that this inconsistency may be due to differences in technique or to the use of old pigment. Nevertheless, it clouds the issue regarding uveopigmentary sensitization.

Woods,[15] in presenting the evidence favoring the allergic hypothesis, admitted that his production of experimental uveitis was open to question, and that while he strongly believed the therapeutic response to uveal pigment was specific, it could not be definitely proved. Examination of the published protocols of Woods's animal experiments, and a review of his reports on the therapeutic use of uveal pigment, indicates that the former showed encouraging but inconclusive results, and that the latter left slight doubt as to its specificity.

However, regardless of objection to certain features in the uveopigmentary sensitization hypothesis, the overall evidence is imposing. This theory readily explains the long incubation period of sympathetic ophthalmia, the failure to find any specific organism, and the rather uniform presence of hypersensitivity to uveal pigment. It also explains the infrequent occurrence of the disease, since only a relatively small number of individuals have an allergic tendency.

Criticisms of allergic theory

On the other hand, several factors are frequently cited in the literature as not harmonizing with this hypothesis, and which indicate that something besides allergy enters the picture. As a matter of fact, not all of these factors necessarily conflict with the allergic theory. An example is the appearance of papillitis as an early and prominent symptom of sympathetic ophthalmia, particularly in cases which show little or no uveal involvement. A possible explanation of the participation of the optic nerve in these cases may be the occasional presence of pigment in its anterior regions.[27]

The dissimilarity of the histologic picture of sympathetic uveitis and that of known allergic reactions has been frequently cited as conflicting with the allergic theory. It is true that the histologic changes appearing in sympathetic ophthalmia resemble those produced by infectious agents. However, as pointed out by Friedenwald,[18] allergic reactions play a greater or lesser part in the histologic changes which take place in infected tissue. Although in some instances the tissues are directly injured by the toxin, in others, such as tuberculosis, an allergic reaction dominates the process, and it is this reaction which determines the characteristic histologic picture. If this is true in tuberculosis, it is conceivable that it may also be true in sympathetic ophthalmia. Further evidence of the similarity of allergic reactions and sympathetic disease is provided by Friedenwald's histologic skin studies, and by the fact that a pathologic picture of allergic origin closely resembling sympathetic ophthalmia has been obtained by injection of horse serum into the opposite eyes of sensitized rabbits.[28]

One of the chief criticisms of Elschnig's original theory was his contention that one of the body tissues was capable of acting as an antigen. This was in conflict with the immunologic rule that an animal cannot be sensitized against his own protein, the "horror autotoxicus" of Erlich. However, in the light of our present knowledge of immunology it is conceivable that the organ specific uveal pigment may have an antigenic action in an homologous animal.

The second phase of this hypothesis has also received much criticism. According to Elschnig, after sensitization has occurred further absorption produces the shocking dose causing anaphylactic intoxication. It has been argued that if this were true there would have to be an interval between the sensitizing and shocking doses in which no antigen was in circulation, on the assumption that the state of anaphylactic shock can occur only when the whole of the sensitizing dose has disappeared.[29] This would mean that uveal pigment must be set free from the injured eye a second time, but not until the pigment produced by the original injury has completely disappeared from the circulation: and this would seem to be a most unusual circumstance to occur during the course of traumatic uveitis. This objection is based on the false premise that there must be an antigen-free period between the sensitising and shocking doses. That this is not true may be demonstrated by such conditions as serum sickness, the positive reaction to tuberculin in active tuberculosis, and many other conditions.

Another argument against the allergic theory is that prophylactic enucleation of the exciting eye would be of no value once sensitization had occurred. Instead, the resultant trauma might be sufficient to act as a shocking dose in causing the disease. To me, this argument does not seem valid. Enucleation of the exciting eye should act to prevent the further absorption of uveal pigment from it and thus tend to prevent the occurrence of the shocking dose. Even admission that the trauma caused by enucleation might stir up the uveal pigment enough to produce it, does not necessarily conflict with Elschnig's

contention. For it would seem to be an unusual circumstance, and it could account for the not infrequent occurrence of sympathetic ophthalmia after prophylactic enucleation.

Factors supporting criticisms

There are two factors which provide quite definite proof that sympathetic involvement is not a purely allergic reaction. These are: first, the fact that sympathetic inflammation does not follow nontraumatic uveitis and other conditions in which there is uveal disturbance; and secondly, the occasional occurrence of hypersensitivity to uveal pigment in patients who do not develop the disease.

The occasional appearance of this "false positive" skin reaction led Woods to modify Elschnig's original theory. His present conception is that the development of allergy to uveal pigment apparently sets the stage for the production of sympathetic ophthalmia by disturbing the immunologic defense mechanism, and that it determines the histologic picture, but that some other unknown factor must be present to initiate the onset of the disease.[20]

The most perplexing feature in this theory is the direct inciting cause. The most generally accepted opinion is that an exogenous infection enters the blood stream and becomes localized in the sensitized uninjured eye. It has been suggested that some unidentified organism or virus may be the infecting agent, or that tubercle bacilli become lodged in the eye, or that the reaction is tuberculo-toxic. However, the whole matter is entirely speculative and it is possible that the inciting factor is entirely nonspecific.[30]

Theory of intraocular melanophores

Friedenwald[18] has suggested the novel theory that the additional factor necessary to initiate the inflammatory reaction is a proliferation of the intraocular melanophores which releases the uveal pigment and makes it available for allergic reaction. He explained his hypothesis as follows:

"In some cases following an injury to the eye, the body becomes hypersensitive to uveal pigment. This alone is not sufficient to elicit the disease, for cells containing melanin granules show no abnormal reaction, in their resting state, to the presence of allergic antibodies. When, however, such cells proliferate, they become vulnerable to the antibodies, and undergo autolysis, releasing their melanin granules, which, in time, elicit the allergic inflammatory reaction."

The pathogenic effect of the melanin granules, and especially of the precursors of melanin, was investigated by Spyratos,[31] who concluded that they act as virulent proteins. However, it is improbable that melanin is the actual inciting agent since it is not thought to contain lipoids or fatty acids[32] which are believed to be essential to the production of the type of granulation tissue found in sympathetic ophthalmia.[33]

Variations of Elschnig-Woods theory

Several variations of the Elschnig-Woods theory have been proposed. Oguchi[34] expressed the belief that liberated chromatophores are responsible for sympathetic inflammation. According to his conception, trauma of the uvea frees immune bodies which attack and destroy the neighboring chromatophores until this change takes place in the entire uvea. The liberated and wandering chromatophores, which act as antigen and as allergen, produce an allergic infiltration when they are in sufficient quantity, or the tissue is electively sensitized. If the liberated chromatophores pass on to the other eye, sympathetic ophthalmia ensues. The pathway may be the blood stream, the optic nerve, or the orbital facial venous system.

Marchesani[35] apparently believes that nonspecific bacterial protein absorbed from the eye produces a paired organ sensitivity of the two eyes, making it possible for the bacteria to localize in the second eye. He based

his conclusions on rabbit experiments in which he produced a histologic picture somewhat resembling sympathetic choroiditis in the fellow eye by repeated intravitreal injections of broth cultures of Bacillus subtilis. However, his conclusions are open to serious doubt, for his assumption that the reaction was allergic has been challenged by Iga[36] and Kiyosawa[37] who, separately, repeated the experiments and concluded that the reaction was due to bacterial metastasis. Their contention receives support from the fact that Friedenwald and Rones[38] found lesions almost identical to those described by Marchesani in eyes of patients who had died of general septicemia.

Riehm[39] reported experiments in which he found that repeated intraocular injections of foreign protein (horse serum) produced sensitivity of the fellow eye in pigmented rabbits, but caused no reaction in albinos. He considered the process to be an elective sensitization of paired organs rather than sympathetic disease. He expressed the belief that sympathetic ophthalmia following perforating wounds was due to an unknown infective agent which acts as an antigen only in human uveal tissue, and sensitizes the uvea of both eyes—an anaphylactic inflammatory reaction to a bacterial antigen. On the other hand, he regarded the reaction as purely allergic, with elective sensitization of the fellow eye, in sympathetic inflammation following such conditions as intraocular melanoma and subconjunctival scleral rupture.

Schlaegel[40] repeated Riehm's experiments but used smaller and fewer doses of horse serum. He found a minimal response in the fellow eye and an equal reaction in pigmented and albino rabbits. However, he did not consider that the results of his investigation had discredited the pigment-allergy theory.

In evaluating animal experiments the biologic and immunologic differences between animals and man must be remembered. Samuels[41] has pointed out the dissimilarity of the rabbit and human choroid. In rabbits, apparently, it is constitutionally more resistant and the circulation more active in carrying away toxins. As a result, irritative lesions cause lymphocytes to appear, but pus cells are notable for their absence. It is not difficult to make a mistaken histologic diagnosis of sympathetic ophthalmia in rabbits, due to the fact that groups of capillaries and endothelial buds in the dense lymphocytic infiltrations may simulate epithelioid cells.

Discussion

Allergy to uveal pigment is only one hypothesis concerning the pathogenesis of sympathetic ophthalmia, but time does not permit discussion of other theories, some of which are built on firm foundations. I have endeavored to review some of the progress and the present status of what up to now is probably the most complete and most carefully worked out theory of this disease. It seems probable that sensitization to uveal pigment plays at least some part in the production of sympathetic ophthalmia but to what extent, time and future studies alone will tell.

Vogt-Koyanagi Syndrome and Harada's Disease

The occasional occurrence of alopecia and poliosis in sympathetic ophthalmia and in other types of uveitis has long been noted. Nettleship, many years ago, suggested that similar pathologic processes were probably present in sympathetic disease and in destructive iridocyclitis with poliosis.

Two types of bilateral nontraumatic uveitis possessing most of the characteristics of sympathetic ophthalmia have been identified. These two conditions are probably related, and according to many authors they comprise a single entity. In one, the Vogt-Koyanagi syndrome, the uveitis is associated with alopecia, poliosis, and, frequently, pigmentary changes of the skin and auditory disturbances. The other milder syndrome, Harada's disease, is characterized by a ser-

ous choroiditis which produces temporary detachment of the retina.

Only a few eyes of patients with the Vogt-Koyanagi syndrome have been examined histologically. These have shown a similarity to sympathetic uveitis, but with the exception of the case studied by Matsuoka[42] there has been an absence of epithelioid cells and giant cells. Because Harada's disease is apparently self limiting, histologic material has been available in only one case in which there was secondary glaucoma necessitating iridectomy.[43] Pathologic examination showed giant cells of the Langhans type. However, the disease has been reproduced in rabbits' eyes by the injection of cerebrospinal fluid and other substances from patients with this condition. The histologic picture resembled sympathetic ophthalmia except for the absence of giant cells.[44]

Because of the histologic and clinical resemblance of these two conditions to sympathetic uveitis, attempts have been made to explain them on an allergic hypothesis. The basis for this idea was Elschnig's[45] contention that alopecia and poliosis in association with sympathetic ophthalmia resulted from the antigenic effect of uveal pigment, the hair being involved because of its richness in pigment.

Peters[46] speculated that the disturbance of hearing in the Vogt-Koyanagi syndrome might be due to sensitivity of the pigment in the basilar membrane of the labyrinth, and Cramer[47] attempted to explain the hair disturbances on the same basis. However, the dearth of pigment in the basilar membrane and the lack of knowledge of its physiologic function caused Koyanagi and others to object to Peters' explanation. Koyanagi[48] explained further, that hearing defects usually occur before uveal pigment can have exerted its antigenic action. Weskamp[49] suggested that it might be possible to explain all of the symptoms of the syndrome by anaphylaxis, since the majority of them originate in tissues rich in pigment. Oguchi[34] impressed by the similarity to sympathetic ophthalmia expressed the belief that the syndrome might be the result of liberated and wandering chromatophores which act as allergens.

Rosen,[50] who is apparently strongly convinced that the Vogt-Koyanagi syndrome and Harada's disease are a common entity and are due to allergy to uveal pigment, suggested that these two conditions probably represent different degrees of involvement in individuals of variable sensitivity. He speculated that in Harada's disease an immunity is soon developed to a severe allergic reaction; whereas, in the Vogt-Koyanagi syndrome the allergic reaction is much less acute, with practically no immune response except when stimulated.

In the four cases of the Vogt-Koyanagi syndrome in which cutaneous sensitivity tests to uveal pigment have been made, a positive reaction occurred in only one.[51, 52] It was assumed that the negative reactions were due to desensitization, as all of these cases had already passed the acute stages when the test was done. However, as pointed out by Rados,[53] this explanation is not entirely adequate since the positive case was also of rather long standing.

It is apparent that an allergic basis for the production of the Vogt-Koyanagi syndrome and Harada's disease is far from established. In spite of the clinical and histologic similarity of these diseases to sympathetic ophthalmia, it is possible that the etiologic causes are entirely different. Beauvieux and Bessiere[54] concluded that rather than indicating a common etiology, it suggests that different viruses may have similar tissue affinities. While it is tempting to speculate that allergy to uveal pigment at least enters the picture, further proof must be provided.

504 State Tower Building.

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THE ETIOLOGY, SYMPTOMATOLOGY, AND TREATMENT OF JUVENILE GLAUCOMA*

ORWYN H. ELLIS, M.D.
Los Angeles, California

It is the purpose of this paper to present the etiology, symptomatology, and treatment of juvenile glaucoma. This condition has been usually regarded as entirely familial; however, I now have five patients between the ages of 15 and 26 years, in whom there is no history of familial tendency. With the introduction of gonioscopy and goniotomy the condition can now be properly diagnosed and operation, when indicated, carried out under direct observation.

To Troncoso and Barkan[1] we owe a debt of gratitude, since it is through them that our knowledge and understanding of glaucoma has been greatly increased with the introduction to ophthalmology of the routine and simplified use of the gonioscopic examination. Now that we can see directly the angle of the anterior chamber, its trabecula, and any obstruction to the outflow of aqueous, our understanding of the filtration angle has been increased. With the gonioscope lens we can view the block of the angle in congenital glaucoma. Likewise, with a clear view of the angle, adult cases can now be classified anatomically into narrow- and wide-angle glaucoma, instead of being classified according to the previous clinical groupings.

ANATOMY OF ANTERIOR CHAMBER

Gonioscopy is so thoroughly an anatomic examination that a review of the anatomy of the anterior-chamber angle is in order. As the cornea passes into the sclera, its inner surface makes an angle with the anterior surface of the iris. Approximately at the apex of this angle in the cornea is normally located the canal of Schlemm. The canal is separated from the anterior chamber by the trabecula, the interstices of which are lined with endothelium. The corneal endothelium, according to Reese,[2] as well as some of its cuticular product, is continued from the cornea to cover the interstices of the trabecula. The structure of a trabeculum, therefore, is a central collagenous fibrous strand around which, as a thin glass membrane, is a cuticular product and this in turn, is covered by the endothelium, which fills the interspaces and/or forms a Descemet's-like membrane along the inner surface of the angle.

With the gonioscope lens in place, as one approaches the anterior-chamber angle from the corneal side, a prominence is first seen which is the anterior border ring of Schwalbe. This constitutes the termination of Descemet's membrane. Between this and the next landmark, which is the white line of the scleral spur, can be seen the individual fibers of the trabeculum running in meridional and circular directions. This zone of Schlemm's canal appears as a faint grayish or occasionally pinkish band. In deeply pigmented eyes, the trabecula is seen to be coated with pigment granules, and the trabeculae then appear dark brown.

As so well described by Barkan,[3] lying directly on this surface there can be seen a few very delicate branching trabecula, which are an extension of the anterior surface of the iris and extend to the posterior corneal surface. These represent that filamentous portion of the network of the angle which anatomists describe as the uveal meshwork. If the iris is removed from the ciliary body in the wet specimen, the anatomist refers to that portion of the meshwork which remains connected to the ciliary body as the scleral mesh, and the very thin portion remaining attached to the iris as the uveal meshwork.

* Presented before the Mexican Association for the Prevention of Blindness, Mexico City, August, 1947.

Teased preparations also show these fibers. Still following around the chamber angle, there is normally seen the dark granular band of the ciliary body, only visible with a wide-open angle. Adjoining this is the anterior surface of the iris.

In juvenile and congenital glaucoma the normal appearance is considerably altered, and none of the usual landmarks can be seen. With the gonioscope lens in place, the anterior chamber is of normal depth, but the angle is everywhere seen to be filled with tissue obstructing the view of even the anterior border ring of Schwalbe. This anomalous filtration angle network is normally present in the fetus until the sixth month and is known as the pectinate ligament. The word pecten means "comb" and refers to the fact that its strands are like the teeth of a comb which has been bent into a circle with the teeth pointing inward. These strands are best developed in horses, seals, and other animals (Walls),[4] where there may be teeth-like processes in several meridians forming a fan somewhat like the situation in reptiles. In horses they are continuous with and identical to the material of Descemet's membrane.

Microscopically in the human eye are seen strands of trabecular tissue or even a mass of considerable density stretching across and blocking the angle of the anterior chamber (Parsons, 1904).[5] This is a persistence of the fetal condition wherein both the sclera and the uveal portions of the trabecula persist, according to Duke-Elder.[6] The etiology of the condition is, therefore, due to the deformities of the angle of the anterior chamber—a persistence of the prenatal or prehuman condition—preventing the free drainage of the intraocular fluids, and thus leading to increased pressure in the eye. This is a developmental abnormality of the mesoderm in the region of the corneoscleral junction. Verhoeff[7] examined two cases of juvenile glaucoma, microscopically, and found in each a defective filtration angle similar to that found in hydrophthalmia.

Hereditary factors

Stokes[8] writes that if it is true that congenital glaucoma (hydrophthalmia) and glaucoma of the hereditary type are essentially the same disease with the same underlying pathogenesis, then the former variety should arise commonly among the offspring of glaucoma patients. There is as yet little evidence that such a sequence is potential. When it does occur it is of interest because it links up the two types.

Lohlein[9] reports a family in which the youngest of the four sisters had hydrophthalmia and the three others had juvenile glaucoma. Scheerer[10] and Korte[11] report families having hydrophthalmia and juvenile glaucoma, and glaucoma simplex with hydrophthalmia, respectively. These cited relationships between the two groups point to an underlying genetic cause leading to a failure (more or less complete) of development of the filtration angle. If the failure is complete, congenital hydrophthalmia results. If it is incomplete, the drainage will be maintained for a variable time, until the sclerosing changes of adult life can no longer be compensated.

Stokes[8] found that when hydrophthalmia is transmitted it appears as a simple recessive; that is, it will not develop unless two conductors of the disease have offspring. Hereditary glaucoma appears as a simple dominant in practically all published pedigrees. Either parent who transmits the disease is usually affected. The chance that an offspring of a glaucomatous person would be similarly affected is in theoretical ratio of 50:50. The chance that an offspring of normal persons would be affected appears to be negligible. Consanguinity does not seem to be a factor.

Age of onset

The age of onset of increased pressure varies in reported pedigrees. The onset of the disease may occur in infancy (hydrophthalmia), early in youth (juvenile glau-

coma), or in adult life. The importance of heredity in glaucoma is as yet not known. Therefore, in these congenital glaucoma types it behooves us to be on the alert for the occurrence of other cases in the family. Keerl[12] analyzed the cases of the University of Leipzig and found heredity to be a factor in 18 percent of cases of juvenile glaucoma. The age grouping was as follows: from 6 to 15 years, 14.8 percent; from 16 to 25 years, 37 percent; and from 26 to 35 years, 48.3 percent. The age of onset in the reported pedigrees varies from 16 to 30 years in families reported by Stokes,[8] Shumway,[13] Calhoun,[14] Courtney and Hill,[15] Bell,[16] and others. According to Allen,[17] 30 years appears to be the upper limit of age of onset.

Regarding the lower age limit for juvenile glaucoma, Anderson[18] uses distention of the eyeball. Thus he writes that due to glaucoma a congenital defect may be classified as juvenile, if it fails to develop until the eye is no longer distensible. The corneal measurements in juvenile glaucoma are all within the limits of normal.

Date of onset

The date of onset of juvenile glaucoma is, in many cases, indeterminate. Thus the reduced age of onset in the younger patients reported could be due to more prompt diagnosis, and must not be taken as a proof of a general tendency to anticipation of the disease, although some anticipation has been reported (Allen[17]). Anderson[18] states that in 87 percent of cases of hydrophthalmia the diagnosis was made before the age of one year.

Cases of juvenile glaucoma are usually chronic; however, the author has seen one patient, aged 26 years, with an acute attack, and a second with a history of a recent acute attack. Allen[17] reports that 18 percent of cases of juvenile glaucoma have acute episodes. He states also that juvenile glaucoma tends to occur more frequently in patients with dark irises.

Relation to myopia

It is still uncertain whether the myopia found in hereditary glaucoma is entirely secondary to the increased intraocular pressure or whether the relation is one of a correlated inheritance similar to that between myopia and other ocular anomalies. According to Stokes,[8] a high intraocular pressure no doubt is a factor in the production of myopia because of the elasticity of the eyeball. The onset of the myopia could even prove compensatory to the high tension changes of glaucoma. Myopia is seldom present in senile glaucoma, but is common clinically in juvenile glaucoma. According to Duke-Elder high degrees of myopia and even hypermetropia are frequently found.

Myopia, according to Parsons[19] is due to the following causes:

1. Decreased corneal convexity.
2. Decreased convexity of lens due to stretching of the suspensory ligament.
3. Displacement of the lens backward—could be due to elastic sclera.

The incidence of myopia in hereditary glaucoma, which averages about 60 percent in all reported families, is sufficiently great to suggest that the hypertension has been influential in producing the bulbar distention and the altered refraction. The occurrence is certainly more than normal and cannot be accidental or incidental.

Influence of race and sex

Stokes[8] found that females are affected a little earlier than males, which is also true of patients with nonhereditary adult glaucoma.

There is no definite racial factor in these cases according to Courtney and Hill.[15] The predisposition of the more nervous or unstable races to acute glaucoma does not seem to hold for the juvenile disease, which is usually of the chronic type.

It is certain that the age limits in glaucoma vary from person to person, as well as in different periods of life. This is even

more true of juvenile than adult glaucoma, and may partly explain the lack of sustained and fixed manifestations of this disease.

Symptoms

By far the most frequent complaint is headache, which is very severe, frequently recurring, usually quite persistent, not relieved by aspirin, present more often in the mornings, and which leaves upon retiring.

Another early symptom, according to McKee,[20] is colored rings around lights—seen first at intervals, then present constantly. Later a sense of fullness or pain in the eye develops. Often these patients give the history that for years they have noticed that their pupils are abnormally large. In the examination, the cornea is usually clear and lustrous; or rarely, if the tension is high, it is steamy. The anterior chamber is always deep, and the pupils are moderately dilated. The disc is usually normal, but may appear slightly pale. No cupping has been seen by me.

Visual fields

The visual fields often show contractures resembling adult glaucoma.[21] The intraocular pressure is rarely found to be elevated, but shows considerable fluctuation, often varying 10 mm. Hg (Schiøtz) from examination to examination. A high intraocular pressure finding is the exception rather than the rule, thus the diagnosis lies entirely with the use of the gonioscope lens. In megalocornea a large expanse of ciliary body is seen, and the normal appearing drainage angle is clearly visualized. This is in contrast to the juvenile glaucoma angle in which the landmarks are covered with dense tissue.

Duke-Elder[6] stated that the condition could stabilize itself in early youth, which could be explained by a stretching process opening up the trabeculae of the angle of the anterior chamber, or by the atrophy of the persistent embryonic tissue in this region. However, this tissue, in most cases, produces embarrassment to drainage.

Verhoeff[7] stated that in juvenile glaucoma the outflow was not completely cut off, at least at first, and, therefore, in early life there is not enough increased intraocular pressure to enlarge the globe; whereas, in hydrophthalmia the pressure is sufficient to do this. Different grades of hydrophthalmia are often present in the same patient, as the condition in one eye may be considerably more advanced than in the other eye. Thus juvenile glaucoma and hydrophthalmia could simply represent differences in degree of the same condition, and Lohlein stated that juvenile glaucoma is a form of hydrophthalmia in which the congenital anomaly of the chamber angle is not sufficient to produce a full-blown hydrophthalmia.

Case reports

At this time I wish to present the case histories of three patients which are in addition to the cases of two patients, aged 15 and 26 years, previously reported.

Case 1

History. M. Q. K., a Chinese physician, aged 26 years, was first examined on December 20, 1946, with the history of an acute attack of glaucoma in the right eye in April, 1945. The present attack began three hours previously with pain, headaches, halos, and blurring of vision of the right eye. He had started using miotics intensively since the onset. There was no history of glaucoma of any type in the family.

Examination. O.D.: Vision, uncorrected, was counting fingers at 3 feet, corrected to 20/70 with a −8.25D. sph. ⌒ +1.25D. cyl. ax. 85°. Tension was: 25 mm. Hg (Schiøtz). The disc showed good color. Fields were normal. O.S.: Vision, uncorrected, was counting fingers at 3 feet, corrected to 20/25 with a −8.0D. sph. ⌒ +2.0D. cyl. ax. 93°. Tension was 28 mm. Hg (Schiøtz). The disc showed good color. Fields were normal.

Gonioscopic examination. The angle was everywhere seen to be blocked with tissue

and very heavily pigmented. No normal angle landmarks were visible.

Diagnosis. Juvenile glaucoma, bilateral, subsiding acute attack in the right eye.

Operation. Under morphine-hyoscine anesthesia and novocaine, locally, a goniotomy was performed as described in a previous paper.[21] More than one fourth of the circumference of the trabecular area was incised on the nasal side of the right eye.

Note. Eight months after this operation an additional acute attack occurred with subsequent goniotomy. Three months afterward a fourth attack of glaucoma suddenly appeared, again following an entirely normal intervening period. A more extensive goniotomy was done following which the tension has remained below 20 mm. Hg. The patient is using miotics only at bedtime. Between the 3rd and 4th operations on the right eye, a goniotomy was done on the left eye as a prophylactic procedure since the tension had occasionally risen above 20 mm. Hg and the gonioscopic appearance of the angles of the two eyes were identical. The corrected vision remains 20/20 in each eye with full fields and normal size of blindspots.

This patient has now been under observation for six months. The tension averages 16 mm. Hg (Schiøtz) in each eye. There have been no further glaucoma symptoms. Premature presbyopia is present, requiring a +1.0D. sph. reading addition.

Case 2

History. B. O., a white girl, aged 19 years, was first examined on March 8, 1947. She complained of very severe headaches for the past five years. There had been occasional blurring of vision of the right eye for the past two years; however, recently the blurring had been followed by intense headaches. For the past three months the headaches occurred daily. She had never noted any halos. There was no history of glaucoma of any type in the family.

Examination. O.D.: Vision, uncorrected, was 10/200 corrected to 20/20 with a −2.5D. sph. Tension was 21 mm. Hg (Schiøtz). The disc showed good color and the fields were normal. O.S.: Vision, uncorrected, was 10/200 corrected to 20/20 with a −2.25D. sph. Tension was 19 mm. Hg (Schiøtz). The disc showed good color and the fields were normal.

Gonioscopic examination. In each eye, about one third of the circumference of the chamber angle was seen to be occluded with tissue, and none of the normal landmarks could be seen in that area. The rest of the angle was normal with only scattered areas of pectinate ligament occluding the angle.

Course. On a later visit the tension, after using prostigmin, was: O.D., 10 mm. Hg; O.S., 19 mm. The patient was continued on prostigmin (2½ percent) twice daily in each eye, and the tension averaged 16 mm. Hg, examined at different times of the day. One headache occurred three weeks after the original examination. Since then there have been no further headaches and no blurring of vision. She has been followed for five months without any recurrence of symptoms.

Case 3

History. M. L. B., a white girl, aged 15 years, was first examined on February 8, 1947. She complained of headaches and dizziness for the past year. Recently the vision had been blurred in the left eye, and there was also pain over the left eye which had been accompanied by vomiting. There was no history of glaucoma of any type in the family.

Examination. O.D.: Vision was 20/30, not improved with any lens. Tension was 16 mm. Hg (Schiøtz). The disc showed good color. The blindspot was slightly enlarged; the periphery was full. O.S. Vision was 20/25, not improved with any lens. Tension was 19 mm. Hg (Schiøtz). The disc showed good color. The blindspot was about twice normal size; the periphery was full.

Gonioscopic examination. The nasal half of the circumference of each eye was filled

with tissue, and scattered areas were present in the temporal portion.

Course. Prostigmin (5 percent) was prescribed to be used morning and night in each eye. She returned in one week without any ocular complaint, not even headache, and she has been comfortable since the first visit. The vision is 20/20 in each eye and there is no refractive error. The tension has averaged 14 mm. Hg in each eye. The blindspots have returned to normal size and there have been no further glaucoma symptoms. The medication was reduced to prostigmin (2½ percent) at bedtime.

Comment

In one patient with acute glaucoma, the eyes have remained normalized following bilateral goniotomies. The two other cases, both young girls, have maintained normal tensions and have been relieved of symptoms with the use of prostigmin. Vision improved in one case and the blindspots returned to normal size. This recovery of field loss was also present in the two previously reported cases, both patients having maintained normal intraocular pressure following bilateral goniotomies.

Confirming Barkan's recommendation, I have repeatedly found that, in the treatment of juvenile and congenital glaucoma, prostigmin is the drug of choice and that pilocarpine and eserine are regularly without effect in normalizing intraocular pressure. Postoperatively in one patient,[21] prostigmin caused an elevation of pressure, and Allen[17] reports a similar case, also postoperatively.

Discussion

When the diagnosis of glaucoma has been made, especially in the juvenile and congenital types, careful family history and investigation should be carried out, as the condition can be definitely diagnosed with gonioscopic examination. In none of my cases was there any familial history of glaucoma. One must be constantly on the lookout for juvenile glaucoma in young adults, especially in myopic individuals who complain of severe headaches. It is to be noted that two of these three patients were myopic. Often a history of blurred vision with the headaches is obtained.

I have been unable to find previous reports of the successful use of goniotomy in juvenile glaucoma. It would seem to be indicated in cases with acute rise in intraocular pressure, or in patients in whom symptoms and/or tension is not completely controlled with prostigmin.

No report of the gonioscopic examination of the angle in juvenile glaucoma was published before Allen, in 1942,[17] described fine iris processes with fingerlike or weblike projections extending anteriorly toward the limbal portion of the cornea.

As Reese[2] has stated, the corneal epithelium is capable of producing a cuticular product or glass membrane as exemplified by Descemet's membrane. The endothelium retains its ability to produce the cuticular product throughout life, but does not manifest this potentiality except under certain provocations.

For instance, as a sign of senescence, excrescences of Descemet's membrane are frequently produced in the periphery, as Henle's warts; also, as a senile change, Descemet's membrane may become thickened. The excrescences and the thickening may occur in an aggravated form over the entire membrane and produce the conditions of cornea guttata and dystrophia endothelialis corneae.

We also know that a variable amount of pectinate ligament may be congenitally present in eyes. Therefore, is it not safe to assume that in some adult eyes, with the angle partly filled with remnants of embryonic tissue, the deposition of the cuticular produce as a senile change overcomes the final pressure compensatory mechanism in an already somewhat embarrassed angle, and glaucoma results? The formation of the cuticular product thus is the secondary change super-

imposed on a congenital malformation.

Or, might it not be entirely possible that in other cases this tissue in the chamber angle in an adult eye suddenly contracts producing a large area of narrow or even occluded chamber angle, thereby causing embarrassment of the drainage system? Certainly, this is not the answer to the etiology of many cases in which narrow-angle glaucoma occurs. In some instances we can follow the formation of adhesions with the gonioscopic lens. Any glaucoma, Reese states, with increased pressure, will develop anterior synechias, and thus the later stages, which are the ones usually studied pathologically, could entirely mask the primary condition.

Anderson,[18] in 1939, stated that no absolute distinction can be made to separate juvenile glaucoma from hydrophthalmia (congenital glaucoma), on the one hand, and senile glaucoma on the other. It is probable that at one end of the scale—that is, in early life, an innate defect in structure was the cause of hypertension and that degeneration was merely a secondary phenomenon. At the other end of the scale, raised tension could be due, almost entirely, to various degenerative changes associated with old age, and inherited defects would play a minor part.

There could be then an intermediate group in which both factors would be at work in varying degrees. One wonders then how much of the shallowness of the anterior chamber seen in congestive glaucoma and in glaucoma of old age is due to secondary inflammatory and degenerative changes and is, therefore, a sequel to rather than a cause of increased pressure.

I feel certain that juvenile glaucoma is due to the same etiologic condition as congenital or infantile glaucoma; that is, a congenital remnant of pectinate ligament blocks the anterior-chamber angle and thereby produces increased pressure. That varying amounts of pectinate ligament do occur congenitally and persist in eyes is well known. Therefore, it is logical to conclude that juvenile glaucoma cases are those in which congenital tissue remnants are present, but in which the tension had remained normalized or nearly so for years, at least to adolescent life. At this time, some, as yet unknown, event occurs and the chamber angle becomes blocked, or at least partly so, and glaucoma ensues. By this time, of course, there can follow no globular enlargement as the tissues are now no longer distensible. In the adult with congenital tissue remnants of smaller amounts, it would be safe to assume a similar mechanism could be at work with the same end result, glaucoma.

Although my patients have shown no familial tendency to glaucoma, most cases of juvenile glaucoma have been reported as hereditary glaucoma with long family histories. The outcome has heretofore been almost universally poor with conservative or operative treatment. With the reintroduction of goniotomy and the utilization of prostigmin, the outlook has considerably improved. I now have five cases with tension completely normalized.

The technique of goniotomy was reported by Barkan[22] and modified by me[21] with the introduction of a new direct-contact operating lens. Using this method, an incision can be made into the area of the trabeculum under direct observation. Good results were obtained in both juvenile and congenital glaucoma when the operation was performed early in the disease, while the glaucoma was still in the compensatory stages; that is to say, before irreversible secondary anatomic and pathologic changes had occurred.

Thus we now know the underlying etiology, symptomatology, and treatment of this disease. The only gap in our knowledge is the full understanding of the factor that finally upsets the compensatory mechanism and produces the actual onset of the condition.

The eyes of youth ordinarily contain an adequate pressure compensatory mechanism

but once that compensatory mechanism fails, secondary anatomic changes result, which in turn lead to further rises in tension and an amazingly rapid progression of all features in this disease.

The term juvenile glaucoma has been comparatively rarely used in the literature, and this paper is a plea for the clearer understanding and proper recognition of these cases. With the increased, widespread use of the gonioscope lens these patients will receive the correct diagnosis and early treatment, thereby preventing acute attacks in most cases, with the result that many eyes will be saved from blindness.

Summary

Juvenile glaucoma is due to a congenital remnant of pectinate ligament, which can be clearly seen with the gonioscopic lens, blocking and covering over the angle of the anterior chamber, the same tissue that causes hydrophthalmia or congenital glaucoma. Its possible relationship to adult glaucoma is discussed. Goniotomy and/or prostigmin have been found to control this condition adequately. No familial tendency to glaucoma in the cases herein presented was observed. A plea is made for early recognition of this disease.

535 West 6th Street (14).

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SYMPATHETIC NERVOUS SYSTEM AND INTRAOCULAR PRESSURE*

NORMAN S. JAFFE, M.D.
Brooklyn, New York

Within recent years much effort has been exerted in implicating the sympathetic nervous system in the causation of glaucoma. It has been known that alterations in the sympathetic pathways will produce fluctuations in the intraocular pressure.

At one time cervical sympathectomy was the vogue in the treatment of glaucoma.[1] Some have claimed success with irradiation of the cervical sympathetic ganglia. Bloomfield[2] showed that the aqueous of the eyes of glaucomatous patients showed a "parasympathetic deficiency" when exposed to an isolated frog's heart. This may be termed a "sympathetic excess." Schoenberg and Lowenstein[3] have attempted to show with the aid of pupillography that central autonomic structures are involved in glaucoma. Demonstration of this derangement has proved to be of considerable value in the early detection of glaucoma.

It is well known that an attack of glaucoma may be initiated by some emotional upset. Many patients with glaucoma are said to be sympathicotonic. Parasympathetic stimulation and parasympathomimetic agents are efficacious in the therapy. None of the proof may, as yet, be considered undeniably positive. It seems fairly certain, however, that the intimate association between the sympathetic nervous system and intraocular pressure is more than mere coincidence and its role in the etiology of glaucoma, although vague at present, is especially formidable.

The sympathetic nervous system may alter the intraocular pressure in any one of three ways:

1. Effect on general blood pressure.
2. Effect on the minute intraocular vessels.
3. Effect on the unstriped palpebral, ocular, and orbital muscles.

Sympathetic nervous stimulation combined with the subsequent hormonal mechanism (adrenalin) will cause an increase in blood pressure. However, associated with this there is a marked generalized vasoconstriction. Some regions respond with a more profound constriction than others. The skin vessels represent the ultimate in this vasoconstriction. For some reason the ocular vessels exhibit a relatively weak response and consequently offer a poor barrier to the increased tension in the general vascular bed. This accounts for the initial passive rise in intraocular pressure as a result of an increase in general blood pressure.

However, after more prolonged or pronounced sympathetic stimulation the blood-adrenalin level increases still further. Vasoconstriction now becomes effective and the intraocular pressure begins to fall. Thus the pressure will not fall until the intraocular arterioles respond to the increased circulating adrenalin.

The effect on the unstriped musculature must also be considered. It is certainly true that this factor assumes more prominence in an experimental animal, such as the cat, than in a human being. Nevertheless, the effect of stimulation or paralysis of Müller's palpebral muscle in a human being is not without important consequences concerning variations in intraocular pressure. The greater development of the smooth peribulbar musculature and the unstriped muscle in the floor of the orbit in the experimental animal make this consideration more prominent in alterations of ocular tension due to sympathetic nervous-system factors.

In studying the effect of adrenalin on the intraocular pressure, Duke-Elder[4] devised a method of divorcing the arterioles of the eye

* From the Department of Ophthalmology, Jewish Hospital of Brooklyn. This work was supported by a grant from the George Beldock Foundation.

from the effects of the generalized increase in blood pressure. He used the perfused head of a laboratory animal. He noted that small doses of adrenalin (1:10,000) caused an increase in intraocular pressure due to capillary dilatation. Larger doses (1:1,000) produced a variable effect. There was a fall due to effective vasoconstriction or a rise due to contraction of Müller's muscle. The latter usually occurred first and was relatively short lived. The vasoconstriction was more prolonged and consequently there was a drop in tension. It is repeated that these are the effects when the influence of the general blood pressure is made nonexistent.

It has been shown by many [5-8] that stimulation of the cervical sympathetic in animals will cause a sharp rise in intraocular pressure due to smooth muscle contraction. This is followed by a fall in pressure due to vasoconstriction. Section of the cervical sympathetic, on the other hand, has not been so clear cut. Some[9-11] have said there is a fall in pressure; others,[12] a rise. At any rate, it may be safely stated that the sympathetic tonus regulates the pressure of the eye in such a way that factors which ordinarily influence the tension will exert a more marked effect when the sympathetic tonus is eliminated.

Adler, Landis, and Jackson[13] showed that increases in general blood pressure cause an increase in intraocular pressure in the normal eye and a more marked increase in the eye devoid of its sympathetic supply (cervical sympathectomy). They refer to this as the "protective action of the cervical sympathetic." Adler[14] states that after cervical sympathetic section there is a transitory dilatation of the ocular blood vessels. He attributes the fleeting nature of this response to an increased sensitivity of the denervated blood vessels to the adrenalin normally present in the blood. It is probable that the dilatation is transitory but it can hardly be due to sensitization since the law of denervation does not manifest itself for several days after severing the nerve supply.

Adler remarks further that after section of the sympathetic it might be supposed that the intraocular pressure would be increased due to the initial dilatation, yet it is almost impossible to demonstrate this in the experimental animal. In addition, Adler points out that in human beings, after the nerve has been injured, there usually results a lasting diminution in the tension together with the other commonly observed ocular signs (miosis, ptosis, and so forth). He attributes the hypotony to the loss of tone in the unstriped muscle around the globe.

Experimental procedures

The following experiments demonstrate the intricate relationship between the sympathetic nervous system and intraocular pressure.

Experiment 1

The intraocular pressure was measured in each eye of seven normal cats. Four cats were subjected to right cervical postganglionic sympathectomy and three cats to right cervical preganglionic sympathectomy. In each case the tension was measured immediately after the operation. It was also recorded at 12-hour intervals during the first postoperative day and then at daily intervals for the next three days. Each cat showed an immediate postoperative Horner's syndrome. In no instance was the law of denervation[15] manifest during the four postoperative days. This means that there was no exaggerated response to adrenalin, excitement, and so forth. The results are shown in Table 1 which shows that in all cases the intraocular pressure on the operated side dropped immediately postoperatively. The fall ranged from 5 to 7 mm. Hg. The average drop in tension was 6 mm. There was no significant change in the tension in the left eye (unoperated side).

However, in all cases there was a rise in pressure back to the preoperative level. In almost all cases the return to normal was completed by the second postoperative day.

TABLE 1

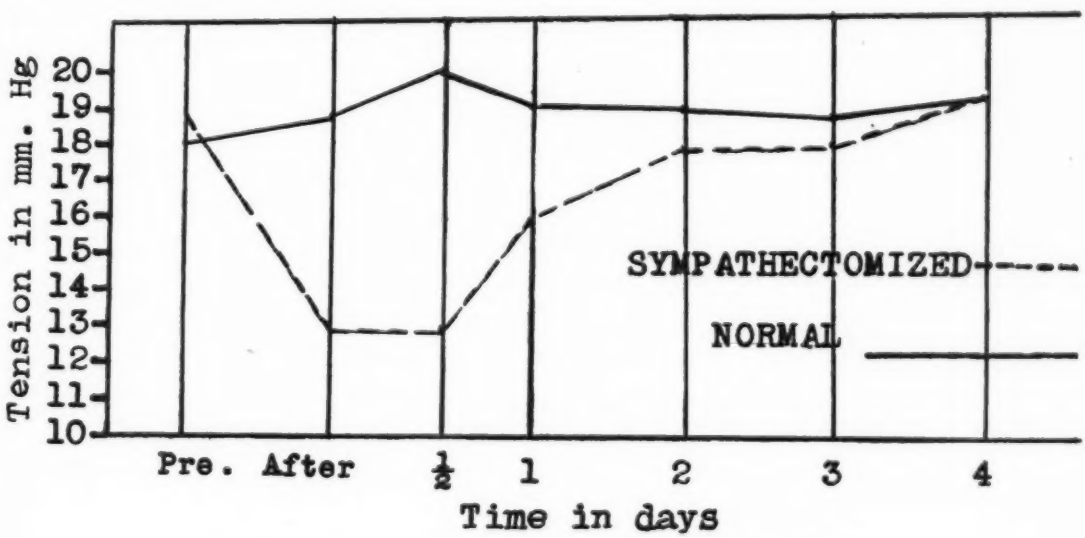
EFFECT OF RIGHT CERVICAL SYMPATHECTOMIES ON INTRAOCULAR PRESSURE OF CATS

Cat	Operation	Preoperative Tension O.D./O.S.	Postoperative Tension					
			Immediate	Days ½	1	2	3	4
24	Postgang.	20/18	15/20	14/20	17/20	20/20	20/20	19/20
25	Postgang.	18/18	11/20	12/20	15/20	15/20	17/17	17/17
26	Postgang.	17/17	12/18	12/20	16/18	18/18	17/17	18/19
27	Postgang.	20/20	13/20	15/20	15/18	20/20	20/20	20/20
28	Pregang.	18/17	11/18	12/18	16/17	17/17	18/18	18/18
29	Pregang.	19/18	14/18	14/19	17/19	19/19	18/19	19/19
31	Pregang.	19/19	13/20	15/20	14/22	18/20	18/19	20/19

During the subsequent two days there was no variation. Graph 1 is a composite record of these results.

EXPERIMENT 2

Seven cats, which had been subjected to right cervical sympathectomy 2 to 4 months before the present experiment, were used. Four of the cats had postganglionic sections and the other three had preganglionic sections. In every case a Horner's syndrome was readily apparent. There was no regeneration in any of the cats, since the iris of the postganglionic sympathectomized animals still showed an exaggerated response to excitement while the preganglionic sympathectomized cats showed a less marked, but exaggerated response.[16]



Graph 1 (Jaffe). A composite record of the results in Experiment 1.

TABLE 2

INTRAOCULAR PRESSURE IN CATS TWO TO FOUR MONTHS AFTER SYMPATHECTOMIES

Cat	Operation	Tension O.D./O.S.
20	Pregang.	20/18
15	Pregang.	20/18
21	Pregang.	20/20
16	Postgang.	18/20
18	Postgang.	15/20
22	Postgang.	18/20
9	Postgang.	16/20

Intraocular pressure was measured in both eyes of each of the seven cats. The results are shown in Table 2.

It is seen that the cats which had a preganglionic sympathectomy showed a normal or a slightly increased tension on the operated side; whereas, those subjected to a postganglionic sympathectomy exhibited a lower tension on the operated side.

EXPERIMENT 3

The seven cats used in Experiment 1 were used here. The present experiment was performed in each case more than three weeks postoperatively so that the sympathetically denervated right iris would exhibit the phenomenon of denervation—that is, extreme sensitivity to stimuli which cause adrenalin liberation.

The tension was measured in the two eyes. Then ether was administered to the animal until it passed through the excitement stage (stage II) and into plane ii of the third stage of anesthesia. The ether was discontinued and the tension was taken. It was then recorded every 15 minutes for two hours (table 3).

The results in Table 3 show that the excitement effect of ether anesthesia caused an initial rise in pressure in all animals. The tension then approached normal at the end of 15 to 30 minutes. After this time it continued to fall. This fall was completed at the

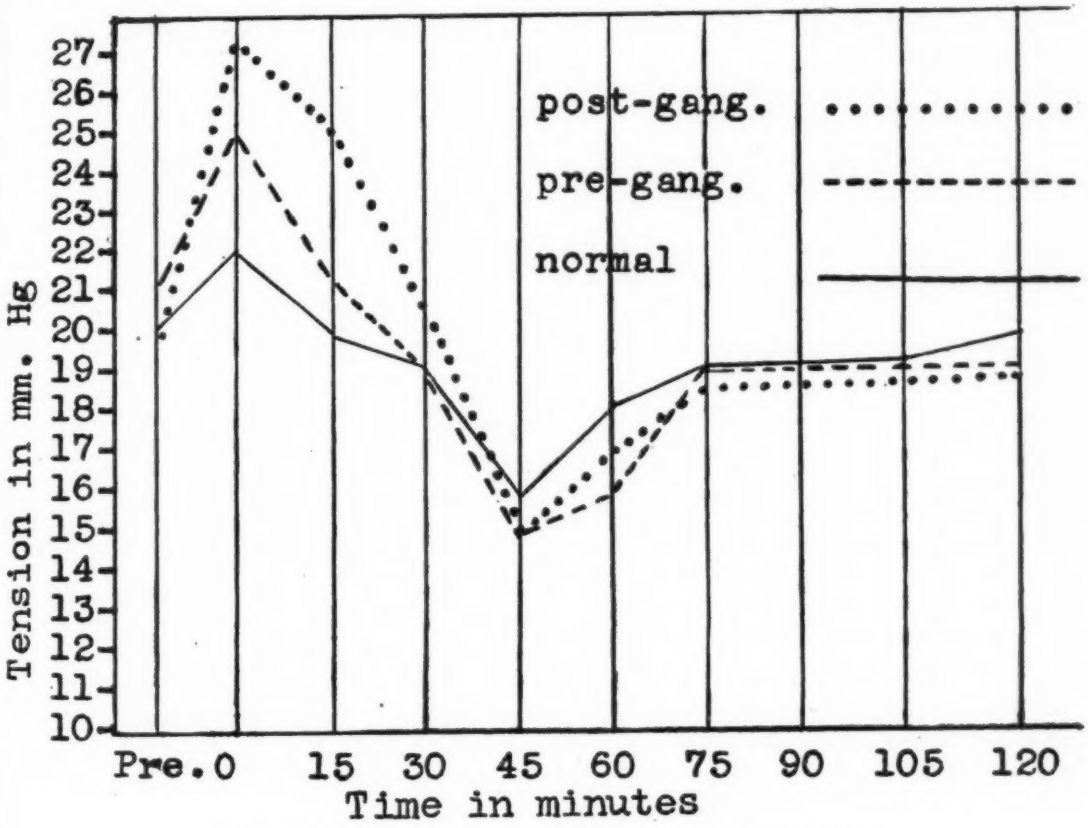
TABLE 3

INTRAOCULAR PRESSURE IN CATS AFTER ADMINISTRATION OF ETHER

Cat	Operation	Tension before anesthesia O.D./O.S.	Tension after anesthesia O.D./O.S.								
			0′	15′	30′	45′	60′	75′	90′	105′	120′
24	Postgang.	16/18	24/21	21/21	18/18	14/17	14/16	18/18	18/18	18/20	20/20
25	Postgang.	18/21	24/21	18/18	18/18	14/16	16/18	18/18	18/16	18/18	18/20
26	Postgang.	18/20	26/24	24/21	21/21	16/18	18/21	20/20	20/20	20/18	20/20
27	Postgang.	18/20	26/24	24/21	21/21	17/17	16/20	18/20	20/20	18/20	18/20
28	Pregang.	18/18	28/20	26/20	20/20	16/16	18/20	20/20	20/18	20/18	20/20
29	Pregang.	20/20	26/24	24/18	18/18	14/16	18/18	20/20	18/20	20/20	20/20
31	Pregang.	18/18	26/21	24/21	20/20	14/14	16/18	18/20	18/20	20/20	20/20

end of one hour when the tension was essentially the same as that recorded before the commencement of ether administration. The second hour (60′ to 120′) showed no variations. The normal eye showed the same response; namely, initial rise with subsequent fall in tension, but to a less exaggerated extent (graph 2).

However, the type of sympathectomy is important after the law of denervation has manifested itself (two weeks or more after the operation), as shown in Experiment 2. The effector cells in the walls of the blood vessels show a more marked sensitivity to adrenalin in the case of a postganglionic than a preganglionic sympathectomy. This may



Graph 2 (Jaffe). A composite record of the results in Experiment 3.

Comment

The drop in pressure following cervical sympathectomy is not permanent. It lasts less than two days. It seems likely that this fall is due to paralysis of the unstriped musculature of the lids, globe, and orbit. The secondary return to normal may be the result of the accompanying vasodilatation caused by the elimination of the sympathetic tonus. There is no difference in the drop observed with preganglionic and postganglionic sympathectomies since both cause a complete removal of sympathetic tonus.

explain why the tension is lower after a postganglionic sympathectomy, since vasoconstriction results when the sensitized effector cells are exposed to the normal adrenalin level. The normal values seen after preganglionic sympathectomy are certainly due to a lack of sensitization of the effector cells.

In Experiment 3, ether, which is sympathomimetic,[16] was given to each animal. Excitement caused both sympathetic nervous system stimulation and adrenalin secretion. The initial rise in pressure of the denervated as well as the normal eye is due mainly to

elevation of the general blood pressure with passive rise in the pressure within the ocular arterioles. Contraction of the smooth musculature also contributes to this initial rise.

The rise is considerably greater with the postganglionic than the preganglionic sympathectomized animals in accordance with the law of denervation. Sensitivity of denervated autonomic structures is twice as great if a postganglionic section rather than a preganglionic section is performed.[17] Sensitivity of the effector cells in the smooth musculature of the lids, globe, and orbit cause this effect. There is also an appreciable amount of sensitization after a preganglionic sympathectomy so that the increase in tension here is greater than in the normal eyes. Also since vasoconstriction is not effective at first in the ocular arterioles and since the sympathetic tonus has been removed, the passive rise in the sympathectomized eye is greater than in the normal eye.

However, after several minutes the tension begins to fall precipitously. This is due to the onset of effective vasoconstriction of the arterioles within the eye. The latter are now able to present an adequate barrier to the induced generalized hypertension. As expected, the normal eyes do not respond as markedly as the sympathectomized eyes. The fall is greatest with the postganglionic sympathectomized eyes since the vasoconstriction is most effective. The preganglionic eyes also exhibit some sensitivity (about 50 percent) so that the fall is greater than in the normal eyes. Again the phenomenon of denervation forms the basis of these results.

As the increased adrenalin level induced by the ether excitement begins to fade, the vasoconstriction wears off and the tension returns to normal. It has been shown that the amount of adrenalin liberated during the excitement stage of ether anesthesia requires about one hour to be metabolized.[16] After slightly more than one hour, the tension in all eyes is about the same as it was before the experiment. This corresponds to the return of the adrenalin level to normal with subsequent release of the vasoconstriction. There are no changes in tension during the second hour of the experiment.

It is clearly shown that at least one of the factors associated with changes in the intraocular pressure is the sympathetic nervous system. Animal experimentation is not perfectly comparable to human experimentation due to differences in the anatomic structures concerned with the maintenance of normal tension. Nevertheless, sympathetic elements are important. However, before its role in glaucoma can be accurately estimated, considerable human experimentation will be necessary.

Summary

Section of the cervical sympathetic nerves in a cat causes a drop of from 5 to 7 mm. Hg in intraocular pressure. This fall is maintained for at least two days after which the pressure returns to normal.

After several weeks, animals previously exposed to a postganglionic sympathectomy will show a decreased tension; whereas, those exposed to a preganglionic sympathectomy will show a normal or slightly increased tension.

Excitement induced by ether causes an initial rise in intraocular pressure in all animals. The rise is greater in a postganglionic than in a preganglionic sympathectomized eye. The rise in the latter is greater than in the normal eye. The increase lasts about 15 to 30 minutes. After this time, there is a fall below normal and then a return to normal tension.

The underlying factors concerned with these changes are discussed.

The sympathetic nervous system is intimately concerned with the maintenance of intraocular pressure and only after much more experimentation will we be able to determine its exact role in glaucoma.

86 Vernon Avenue (6).

Indebtedness is expressed to Dr. Isadore Givner for valuable suggestions and criticisms.

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SULFATHIAZOLE SENSITIVITY: WITH A REVIEW OF THE LITERATURE

ARNOLD A. ALBRIGHT, COL. (MC),* AND
EDWARD L. SERETAN, MAJ. (MC)†
Battle Creek, Michigan

Sulfathiazole sensitivity in its minor manifestations is well known and not infrequently encountered. However, extremely severe reactions simulating Arthus's phenomenon may occur, as indicated by a recent case at Percy Jones General Hospital.

CASE REPORT

History. A former infantry lieutenant, aged 32 years, sustained a grenade wound in action with the Japanese on March 24, 1945. Fragments penetrated his shoulders, right arm, back, left leg, and right eye. Immediately after injury, vision in his right eye was reduced to light perception.

All wounds, except those of the eye, were debrided and sulfanilamide powder was introduced. An undetermined number of fragments were removed from the right orbit on Leyte Island in April, 1945. Another fragment was removed in October, 1945, at O'Reilly General Hospital. Vision improved gradually. However, several episodes of sharp pain in both eyes and frequent frontal headaches, relieved by aspirin, remained as his chief complaints. Discharged on a limited-duty status, he was recalled for reëvaluation and was admitted to Percy Jones General Hospital on January 14, 1947.

Physical examination revealed numerous, well-healed scars on the back, both shoulders, right arm, and left leg.

Eye examination. Vision was: O.D., 20/50, J6, correctible under cycloplegic with a +2.0D. sph. ⌒ +0.75D. cyl. ax. 105° to 20/30+2, J2. O.S., 20/20, J1, correctible to 20/15, with a +1.25D. sph. ⌒ +0.5D. cyl. ax. 105°. External examination was not remarkable.

Slitlamp study of the right eye revealed pigment granules in a fluid vitreous. Ophthalmoscopic examination showed a large area of healed traumatic chorioretinitis in the superior nasal quadrant near the periphery.

* Chief of EENT Section, Percy Jones General Hospital.
† Resident in Ophthalmology, Percy Jones General Hospital.

This was circumscribed by pigment. The left eye was normal.

X-ray examination with Sweet localization revealed a foreign body, 1 by 2 by 1 mm. in size, lying 7 mm. above the horizontal plane of the cornea, 9.5 mm. to the nasal side of the vertical plane of the cornea, and 15 mm. back of the center of the cornea, in or near the sclera of the right eye. The Berman locator confirmed the position of the metallic fragment, and at operation on January 24, 1947, with its help the foreign body was found in the sclera and dissected free without difficulty. A small amount (approximately 30 mg.) of 5-percent sulfathiazole in lanolin ointment was placed in the conjunctival sac and a dry dressing applied.

Reaction. Within 24 hours the patient developed a generalized urticaria, edema of the lids and conjunctiva in the right eye. Itching of the face and body became generalized and severe. A macular rash developed over the trunk and extremities. By this time the right eye was swollen almost completely shut, while the left eye began to show similar involvement. The healed scars of the arms and trunk became swollen, formed blebs which erupted, exuded serum, and finally became slightly necrotic. On the day following surgery, the temperature rose to 101.8°F., with a pulse rate of 142 per minute. Two days later both gradually subsided to normal.

Treatment consisted of repeated irrigation of both eyes immediately after the allergic manifestations were noted, and instillation of adrenalin (1:1,000) in both conjunctival sacs. Benadryl (50 mg.) was given orally every four hours. Except for some relief from itching, there was no amelioration of symptoms. The systolic blood pressure dropped to 75 mm. Hg, and was brought back to normal with adrenalin, following which patient developed auricular fibrillation, confirmed by electrocardiogram.

Course. Urticaria and edema of the eyes increased in severity until both eyes were swollen shut. Edema of the right cornea was severe enough to reduce vision to light perception.

After the administration of 9.5 gr. of quinidine sulfate in graduated doses, the heart rhythm and blood pressure became normal. The electrocardiogram was normal on January 26th. Edema and urticaria gradually subsided during the subsequent three weeks, and the patient's eyes returned to normal.

On February 24, 1947, vision with correction was: O.D., 20/30, J2; O.S., 20/20, J1. On February 28th, the patient met a board, which considered him fit for general duty.

Discussion

Many points of interest arise from this case. The necrosis of the former wound areas resembles the phenomenon described by Arthus in 1903. As he inoculated rabbits subcutaneously with repeated doses of horse serum, at different sites, it was noted that the primary site of inoculation would undergo a severe tissue reaction, becoming injected, swollen, and later undergoing necrosis. This is supposed to be peculiar to the rabbit, according to Topley and Wilson.[1] Our patient had been sensitized previously by sulfonamide therapy.

Untold thousands of wounded servicemen were treated by sprinkling their wounds with sulfonamide crystals. Of these a large number were given further sulfonamide therapy in their subsequent care. Therefore, the number of ex-servicemen sensitized to sulfonamide drugs may be high.

Review of literature

In reviewing the literature, we were struck by the paucity of detailed information concerning idiosyncrasy to any of the sulfonamide derivatives when instilled in the eye. Morrison,[2] in 1942, called attention to allergic conjunctivitis from the topical use of sodium sulfathiazole and sulfathiazole, and cited a case of severe allergic blepharoconjunctivitis,

Shaffer, Lentz, and McGuire,[3] in 1943, called attention to the fact that sulfathiazole used locally on the skin is capable of causing cutaneous sensitization, which may manifest itself as a local contact type dermatitis with or without generalized eruption; also that sensitization produced by local application may be elicited by oral administration.

Alvaro[4] reviewed the literature and listed the effects of sulfonamide compounds on the eye. He lists palpebral edema, conjunctivitis, chemosis, scleral reaction, cells in aqueous, iritis, mydriasis, cataract, edema of the retina, reduction of visual fields, retinal hemorrhages, optic neuritis, undefined blurred vision, transient ametropia, changes in accommodation, and heterophorias as the side effects. Most of those listed are as a result of sulfonamide therapy taken orally.

Alvaro[5] also states that sulfonamides are almost innocuous to the ocular tissues when applied locally, and that when local intolerance occurs, it is demonstrated by lid dermatitis and conjunctivitis, which subsides as soon as treatment is discontinued. He recommends the local use of sulfonamides because of easy penetration and tolerance.

Except for the cases cited, local intolerance is mentioned more or less casually, but there have been no cases reported similar to this one. However, various ocular reactions to peroral sulfonamide have been reported. The first report in literature of sulfonamide intoxication was made by Bucy,[6] in 1937, in which a case of optic neuritis developed.

In 1939, Rosenthal[7] noted field changes with angioscotometry, in which there was a narrowing of the vessel shadow induced by sulfanilamide. In 1940, Baker[8] reported retinal hemorrhages in a patient receiving sulfanilamide by mouth.

Bloom, Leech, and Shaw[9] report a case of temporary blindness, associated with retinal hemorrhage and sulfathiazole nephritis in a patient treated by peroral sulfathiazole for pyelitis and cystitis. Goar,[10] in 1942, reported two cases of retinal hemorrhages following ingestion of sulfathiazole. In 1942, Carlbuerg[11] reports four cases with myopia varying from 2 to 4.50D. due to lens changes. Numerous cases of transient myopia have since been reported.

Bellows,[12] in reviewing the literature, mentions optic neuritis, round hemorrhages around swollen arterioles, greenish scleral discoloration, bluish discoloration, temporary blindness, transient myopia, and field changes.

Savitsky and Weitzen[13] report a case of dysmorphopsia associated with blurring of vision in a woman receiving sulfanilamide by mouth. Laval[14] reports a case of paresis of accommodation in a 17-year-old girl after she had received 11½ gm. of sulfadiazine in five days for an upper respiratory infection.

Scott and Sorsby[15] list the following complications to sulfonamide therapy, but do not specify whether the medication was given locally or systemically: Transitory myopia, retinal hemorrhage, optic neuritis, and conjunctival reactions; for example, color changes, cyanosis, and pallor.

Fritz and Kesert[16] describe a case of acute congestive glaucoma following oral sulfathiazole for sore throat.

In a recent editorial in the *American Journal of Ophthalmology*, Cordes[17] sounds a warning against the indiscriminate use of sulfonamides and penicillin. He states that sulfathiazole, locally in even infinitesimal doses, can induce sensitivity. He also calls attention to reports in the literature of individuals, sensitized to one sulfonamide by local use, reacting to another type of sulfonamide after oral administration. Attention is also drawn to kidney damage and anemia. Cordes, with Thygeson, recommends sulfadiazine as being less toxic and less apt to produce sensitivity, and that sulfonamides should not be used longer than two months.

Sodium sulfacetimide (Albucid soluble) merits consideration, inasmuch as Benedict and Henderson[18] observed only one case in 65 sensitive to this drug.

Summary

1. A small amount of 5-percent sulfathiazole ointment was instilled in the conjunctival sac of a wounded officer previously treated with sulfanilamide. The patient underwent a severe local and generalized reaction. Findings included edema and chemosis of both eyes; generalized urticaria and itching; necrosis of wounds formerly treated with sulfonamide; and fall of blood pressure.

2. Treatment of sensitivity reaction consisted of topical adrenalin, benadryl in 50-mg. doses, and sodium-bicarbonate soaks. Edema did not subside in the right eye for three weeks.

3. A reaction similar to that described by Arthus in rabbits was seen in this case.

4. The literature reveals no similar case of sensitivity.

Conclusion

Severe sensitivity reactions may develop with the use of sulfathiazole, and probably with other sulfonamides. Therefore, a word of caution is offered, with the suggestion that a careful history be elicited in the event of contemplated local use in the eye of sulfathiazole or other sulfonamides. It would appear from the literature that sulfadiazine or sodium sulfacetimide (Albucid soluble) would be the sulfa drug of choice.

Percy Jones General Hospital.

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LOCAL ANESTHESIA IN OPHTHALMOLOGY*

WALTER S. ATKINSON, M.D.
Watertown, New York

The purpose of this paper is to review briefly a few local anesthetic procedures that have been found satisfactory over a period of years.

We have all observed that many operative accidents which occur during intraocular operations are due to a forcible contraction of the orbicularis, the rectus muscles, or to a quick movement of the eye, all a result of incomplete anesthesia. Therefore, it is extremely important to produce complete anesthesia, and as painlessly as possible, before beginning the operation.

While the principal aim of anesthesia is to prevent physical pain, it is also important to allay fear and apprehension which may shock the patient as severely as pain. The patient who comes to us for operation has confidence; otherwise he would not entrust his most valuable possession, sight, to us. His confidence though is easily undermined so every act and word in connection with the preparation of the patient for operation should be carefully studied. Usually the less said, the better. A thoughtless remark or trivial act may excite fear and apprehension. Then, confidence, which is so illusive, is difficult to regain.

Patients undoubtedly differ to as great an extent in their mental as they do in their physical characteristics and so should be approached as differently as one would approach the various surgical conditions encountered.

Before operation and during the preparation of the patient and the production of local anesthesia, patients are particularly apprehensive. Their sensibility to pain is greatly exaggerated so that each needle prick seems to be of increasing intensity. It is, therefore, of the greatest importance to induce anesthesia painlessly in order to retain and strengthen the patient's confidence. Any complaint of pain usually indicates the incorrect administration of the anesthetic rather than a defect in the procedure or the anesthetic agent.

Local anesthesia demands and allows deliberation and care in all of the manipulations of the surgeon, and the time during which local anesthesia is produced offers an excellent opportunity to build up the patient's confidence.

SEDATIVES

Sedatives, if used carefully, are a valuable aid and assist greatly in production of a quiet and tranquil patient. But, as we all know, the action of sedatives varies greatly with different patients. In some they increase excitability and may cause nausea and vomiting, so that it does not seem advisable to use any sedative, particularly for intraocular operations, without first ascertaining the action of the drug on the patient. Usually the patient is admitted to the hospital at least one day before the operation. On the first night in the hospital a sedative may be given which acts as a test dose. This allows the operator not only to learn the action of the drug on that patient but, if it acts in the usual manner, gives the patient a quiet, restful sleep, which is excellent preliminary preparation for the operation and may be repeated with comparative safety before operation.

Some of the barbituric acid group, such as pentobarbital sodium and phenobarbital, are popular preoperative sedatives for globe operations as they rarely cause nausea or other undesirable reactions. They also have the added value of counteracting to some extent the toxic action of the anesthetic agent.

Some patients require a larger dose than

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others. Small doses repeated until the desired effect is produced, seem more satisfactory than the routine use of a large set dose. With too much sedative the patient may be as uncoöperative as an acute alcoholic. Elderly persons often take a sedative regularly and, if so, it is advisable to prescribe the one to which they have become accustomed.

As a test dose, pentobarbital sodium (1.5 gr.) with acetyl salicylic acid (5 gr.) has been found excellent for the average adult. If there is no undesirable reaction, pentobarbital (3 gr.) with acetyl salicylic acid (5 gr.) is given one hour before the operation. Only in exceptional patients is it necessary to increase this dose.

Instillation anesthesia

Since the introduction of cocaine, chemists have produced numerous local anesthetic agents, some of which have in a large measure replaced cocaine. While cocaine is still popular, the chief objections to its use are that it dilates the pupil and may influence the intraocular pressure. Cocaine also causes desiccation of the corneal epithelium and may devitalize the cornea which tends to retard healing.

Tetracaine (pontocaine) hydrochloride, a derivative of procaine, is one of the more popular local anesthetic agents used for instillation anesthesia. The chief advantages of tetracaine are: it smarts very little when it is instilled in the eye; its action is rapid and prolonged; the pupil is not dilated; the intraocular pressure is not influenced, nor is the accommodation affected. Tetracaine does not cause desiccation of the corneal epithelium nor does it devitalize the cornea.

All local anesthetic agents probably delay healing of the corneal epithelium to some extent. Gundersen and Liebman[1] found 0.5-percent tetracaine hydrochloride to be the least toxic to the regenerating epithelium. Smelser and Ozanics[2] conducted experiments which confirmed the recognized belief that frequent topical application of anesthetics inhibited cell migration and mitosis. Therefore, prolonged use of any local anesthetic is contraindicated.

Patients are rarely sensitive to tetracaine but, as with atropine and many other drugs used in the eye, a conjunctivitis and dermatitis of the lids may be caused so this possibility should be kept in mind. Larocaine, metacaine, nupercaine, and other local anesthetic agents may compare favorably with tetracaine. However, the use of tetracaine routinely for 15 years has convinced me that it is an excellent local anesthetic agent. Other anesthetics have been tried and compared with tetracaine clinically but none were found to be superior. The usual strength of tetracaine hydrochloride used for instillation anesthesia is 0.5 percent, although some surgeons prefer a 1-percent solution.

Infiltration and block anesthesia

Although there have been many anesthetic agents produced for infiltration and block anesthesia, procaine hydrochloride (novocaine) is still generally conceded to be the anesthetic of choice for injection.

The strength of the procaine hydrochloride used for the injections to be described later is 2 percent with epinephrine hydrochloride (1:1,000) 1 mm. in 5 cc. The amount of epinephrine is increased for the retrobulbar injection when hypotony is desired and to retard the absorption.

Braun[3] states that a 0.4-percent solution of potassium sulphate added to the solution of procaine hydrochloride greatly increases its anesthetic properties and in this concentration produces neither local nor general injury.

Instruments

Syringes. The Luer-Lok syringes, 2 and 5 cc. (fig. 1), have been found excellent for injections around the eye. They have a lock which prevents the needle from coming off during the injection; they are light and are not damaged if placed in a boiling sterilizer.

Needles. Three sizes of needles are desirable: a 27-gauge needle, 12.5 mm. in length,

for subconjunctival injections, intradermal wheals and small subcutaneous injections; a 25-gauge needle, 2 cm. in length, for deeper subcutaneous injections, production of akinesia, and so forth; and a 22-gauge needle, 3.5 cm. long, for still deeper injections, such as retrobulbar injection within the muscular cone and to block the lids. If the point of the needle used for retrobulbar injections is ground down so that the point is rounded, it is less likely to injure the tissues or pierce a vessel (fig. 2).

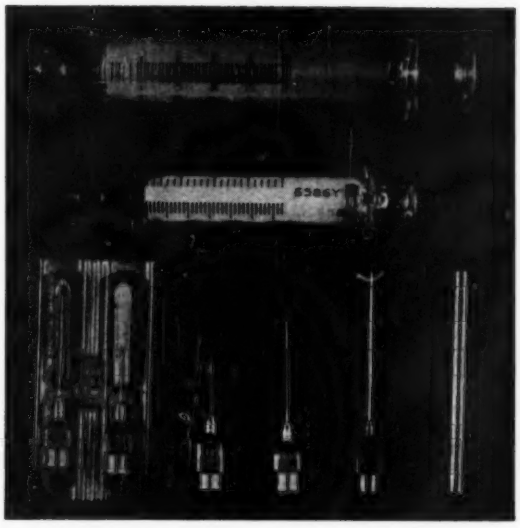

Fig. 1 (Atkinson). Luer-Lok syringes and needles.

Fig. 2 (Atkinson). Rounded needle point and the regular sharp-pointed needle.

Akinesia

The desirability of having a temporary paralysis of the orbicularis muscle for intraocular operations, particularly extraction of cataract, is so generally conceded that comment is unnecessary. The method used to obtain paralysis of the orbicularis seems of less importance than to be certain that it is obtained before the operation is begun. Of the various modifications of the van Lint[4] method of producing paralysis of the orbicularis the simplest and most satisfactory is the one proposed by O'Brien.[5]

As is well known, in the van Lint method the needle is introduced at the temporal margin of the orbit (fig. 3) and an injection is made downward and nasally close to the bone to block the branches of the facial nerve which supply the lower lid. The needle is then partially withdrawn until the point is just beneath the skin and the needle is then redirected and the injection made upward to block the branches which supply the upper lid.

By the O'Brien method the temporofacial division of the facial nerve is blocked just anterior to the tragus of the ear (fig. 3). If the patient is asked to open his mouth, the condyloid process can be easily felt. The needle is then introduced until the condyloid process is just felt with the needle and about 3 cc. of the anesthetic solution is injected. If the periosteum is pricked with the needle unnecessary pain is caused.

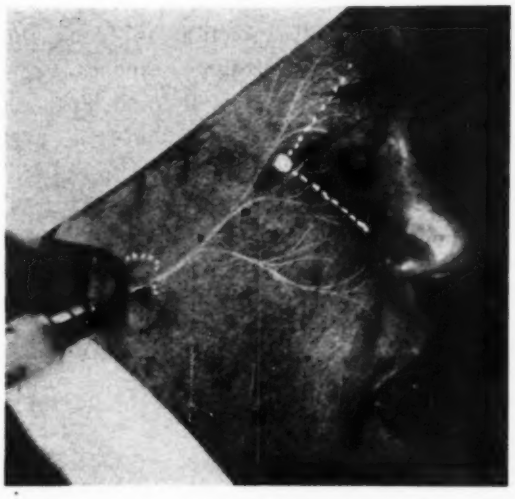

Fig. 3 (Atkinson). The dotted line indicates the site of the van Lint injection, and the needle marks the site of the O'Brien injection, to produce temporary paralysis of the orbicularis muscle.

Deep massage with firm pressure over the site of the injection will diffuse the anesthetic solution and produce more rapid and complete paralysis of the orbicularis muscle. The course of the nerve varies considerably, so that paralysis is not always obtained with the first injection. If, after several minutes, the muscle is still active, another injection should be given and the site of the injection changed to correspond to the more common variations in the course of the nerve. Klein[6] found on dissection that the nerve more often crossed the ramus of the mandible lower than shown in most textbooks. In any event, the object of the injection should be accomplished before proceeding with the operation. A simple test to tell whether or not complete paralysis of the orbicularis has been obtained is to hold the lids open and ask the patient to close the eye. The slightest action of the orbicularis can, in this way, be felt.

One now rarely hears the criticism that the lids may come open under the dressing and so damage the eye. This does not occur, in normal cases, if the dressing is applied carefully. When the eyes are prominent and the lids do not close well, a suture to hold them closed may be easily introduced. The reason the O'Brien method is preferred is because one small injection to block the temporofacial nerve before it branches seems simpler and more efficient than to block its many branches at the orbital margin.

While the principal indication for akinesia is to prevent a "squeeze" or forcible contraction of the orbicularis in globe operations, it is also of value in other operations to obtain a larger operative field when the lids are not held open well, such as operations for detachment of the retina and muscle operations when done with local anesthesia.

When more prolonged paralysis of the orbicularis is indicated, as with patients who have a tic, with spasmodic contractions not only of the orbicularis but of other facial muscles, alcohol may be added to the solution to be injected as suggested by de Saint-Martin.[7]

The following solution, which contains 25-percent alcohol, causes little pain when injected, and the paresis lasts 7 to 10 days, sometimes longer:

Isotonic solution of sodium chloride U.S.P. . .8 cc.
Procaine hydrochloride .....0.2 gm.
Absolute alcohol .....2.00 cc.

Less pain will be experienced if 1.5 cc. of procaine hydrochloride (2 percent) is first injected, after which the syringe may be removed and the needle left in place. Beginning paresis of the orbicularis muscle indicates that the needle is in the proper position. The syringe containing the alcohol is then attached to the needle and 1.5 cc. are injected.

Retrobulbar injection within the muscular cone

In the preparation of the patient for an intraocular operation, such as a cataract extraction, let us assume that the patient has had a sedative and an anesthetic solution instilled in the eye to produce superficial anesthesia of the anterior segment and anesthesia of the conjunctiva. A retrobulbar injection within the muscular cone, or cone injection, is now given to produce more complete anesthesia.

Technique

An intradermal wheal is first made to avoid causing unnecessary pain whenever the skin is to be pierced. To some this may seem unimportant but since the aim of anesthesia is to eliminate pain we should certainly try to avoid causing pain during the production of anesthesia. Braun[8] emphasizes this and reminds us that the skin, with its innumerable nerve endings, is probably the most sensitive tissue in the body.

For the cone injection, the wheal is made just below the inferior temporal margin of the orbit (figs. 4, 5, 6) using a 27-gauge needle with the bevel toward or flat against

the skin. The 22-gauge needle, 3.5 cm. in length, is then introduced through the wheal, the skin is moved upward with the needle until the point just clears the inferior orbital margin. About 0.5 cc. of the anesthetic solution is now injected so that little pain will be experienced when the orbital septum is pierced and it can be pierced more

Fig. 4 (Atkinson). Intradermal wheal.

easily close to its attachment to the orbital margin.

A pause of a few moments allows the anesthetic to work during which time the patient is directed to look upward and away from the site of the injection; in this instance up and to the left for injection of the right cone. The needle is then directed upward and inward, midway between the lateral and the inferior rectus muscles. It is then advanced toward the apex of the orbit for a distance of 2.5 to 3.5 cm., the distance depending upon the size of the orbit. When the needle has reached the proper depth, one should aspirate before injecting the anesthetic solution in order to determine whether or not the needle has entered a vessel. However, in this location this is practically impossible unless there is an anomalous vessel, since here the vessels are normally smaller than the needle and are freely movable.

Again the importance of using a large, 22 gauge, needle not longer than 3.5 cm. with a rounded point should be emphasized. If a 4.5- or 5-cm. needle is used and the point carried to the apex of the orbit, obviously

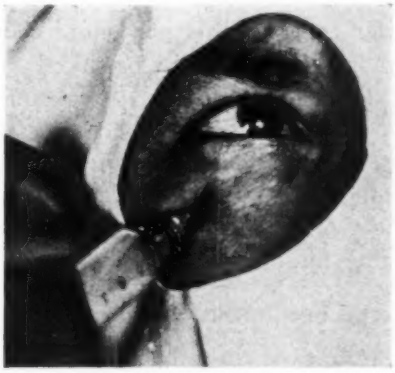

Fig. 5 (Atkinson). Retrobulbar injection within the muscular cone.

a vessel or the optic nerve might easily be pierced, because here the vessels converge to reach their bony exits and are fixed.

From 1 to 1.5 cc. of the procaine epinephrine solution is slowly injected, care being taken not to produce undue proptosis by too large an injection. For the majority of operations on the globe, 1 cc. is sufficient, but for operations on the muscles and for enucleation, 3 cc. or more may be injected, as the proptosis does no harm and, for inflamed

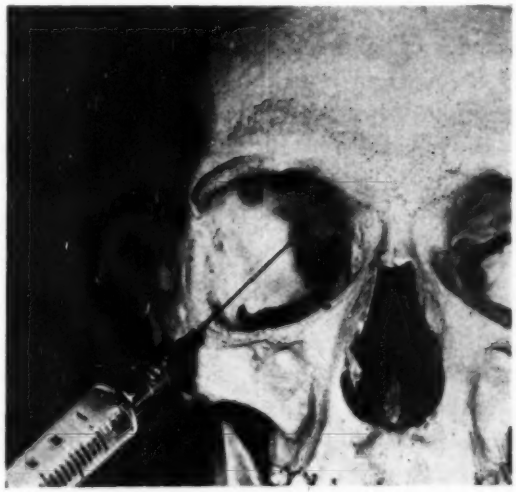

Fig. 6 (Atkinson). Skull showing position of needle for retrobulbar injection.

and painful eyes, more procaine is desirable.

Gentle massage with a rotary motion of the globe causes the procaine solution to become more thoroughly diffused within the muscular cone. In this way more complete and rapid anesthesia is produced. Anesthesia of the muscles is not obtained as rapidly as anesthesia of the globe; so for enucleation and operations in which there is considerable traction on the muscles it is advisable to wait 5 or 10 minutes before beginning the operation.

Blocking of sensory nerves

By means of the injection within the muscular cone, all of the sensory nerves to the eyeball are blocked, and complete anesthesia of the globe is obtained regardless of how inflamed or painful the eye may be. Pressure on the inflamed eye, which before the injection caused severe pain, is not noticed now. The rectus muscles, except the superior rectus, are also rendered practically insensitive to traction.

The injection within the muscular cone also causes paresis of the rectus muscles, except the superior rectus, so that the patient cannot look down easily. Therefore, for such operations as extraction of cataract, it is advisable to use a suture under the superior rectus muscle (bridle suture). With the rectus muscles inactive, little traction is required to hold the eye in the desired position. Also, better fixation of the globe is thus obtained. With paresis of the rectus muscles the so-called squeeze, with loss of vitreous, caused by their contraction is less likely to occur.

The superior rectus muscle usually remains active and may be sensitive to traction. For prevention of pain when this muscle is picked up for the introduction of a bridle suture or for an operation in which there is traction on the muscle, additional anesthesia is necessary. This may be accomplished by an injection of 0.5 cc. of the anesthetic solution along the superior rectus muscle. With the superior rectus muscle active and with paresis of the other extrinsic muscles, there is torsion of the globe, with rotation toward the nose. The torsion is increased when a suture is used under the superior rectus muscle. Therefore, if an iridectomy is to be performed, it should be done from 10 to 15 degrees to the nasal side, so that the coloboma will be at axis 90° when the muscles again function normally. While this is of no particular importance from a visual standpoint, a coloboma looks better in the vertical meridian.

Subconjunctival injections

When the eye is red and inflamed, as in cases of acute glaucoma, a subconjunctival injection should also be given, since the inflamed conjunctiva is not well anesthetized by topical application and only the conjunctiva immediately adjacent to the limbus for a distance of about 3 or 4 mm. is rendered insensitive by an injection within the muscular cone. This is because the nerve supply is from the ciliary nerves, which are within the cone.

The rest of the conjunctiva is innervated by the supraorbital, supratrochlear, infratrochlear, infraorbital, and lacrimal nerves, all of which are outside the muscular cone and are seldom reached by the anesthetic injected within the cone. When the eye is inflamed, less pain is caused if the subconjunctival injection is given after the retrobulbar injection.

For the subconjunctival injection the needle may be introduced through the insensitive conjunctiva directly surrounding the cornea and, if succeeding injections are made through blebs produced by the anesthetic, little pain will be experienced. In this way, pain due to the use of the fixation forceps and to suturing of the conjunctiva is avoided in eyes that are inflamed and painful. A 27-gauge needle is used and slid gently over the conjunctiva until a fold appears in front of the point; the syringe is then rotated, which produces a boring effect. In this way even a dull needle passes easily

through the conjunctiva without the use of forceps. If the bevel of the needle is toward the globe, one is less likely to prick the sclera and cause bleeding.

With such complete anesthesia of the globe, the eye being practically immobile together with paralysis of the orbicularis muscle, operations on the globe can be performed with the same deliberate precision that one can use when operating on the cadaver.

Action of anesthetic

If the patient looks upward and away from the site of the injection, access to the muscular cone is easier. The extension of the fascial sheath of the muscles which closes the spaces between the rectus muscles for a little distance back of the globe is in this way moved forward and upward, out of the way, so that the needle does not strike it, as is shown diagrammatically in Figure 7. If the needle does strike the sheath, pain is caused. The sheath is also somewhat tough, and the needle does not penetrate it easily, with the result that the globe rotates, which to some extent interferes with the introduction of the needle and the equilibrium of the patient.

The injection is made through the skin at the inferior temporal margin of the orbit rather than through the conjunctiva because it is less painful and allows a better angle of approach to the muscular cone. Procaine injected within the muscular cone is apparently held within the cone, since the nerves outside the muscular cone are rarely affected and injections outside of the cone do not reach the nerves that traverse the cone.

How is the solution of procaine held within the muscular cone, since there is no demonstrable fascia between the rectus muscles posterior to the globe, as they converge to their attachment at the apex of the orbit?

Anteriorly, the escape of fluid is prevented by the globe, surrounded by Tenon's capsule, and by the adjacent margins of the sheaths of the four rectus muscles, which are continuous with one another, with a wide sweep between them. In this manner, an intramuscular membrane is formed which, according to Whitnall,[9] exists for a short distance behind the globe. Beyond this the muscles converge to their attachment through the annulus of Zinn, at the apex of the orbit, and

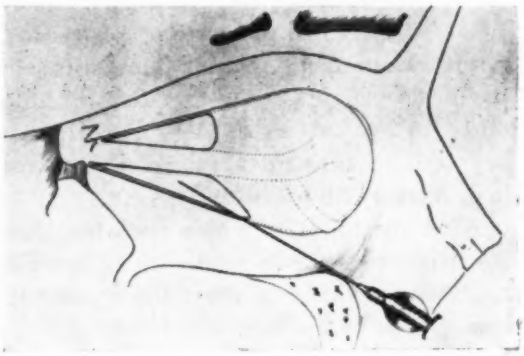

Fig. 7 (Atkinson). Diagrammatic sketch showing the eye turned up and away from the site of the injection, so that the fascial extension is moved forward and upward out of the way.

their margins come closer together, so that comparatively little space is left between them. In this location the escape of the procaine solution is probably prevented, or at least retarded, by the closely packed layer of fat, with its connective-tissue reticulum.

According to Whitnall,[10] the orbital fat which fills the entire space in the orbit not occupied by other structures varies in consistency in different regions. Within the muscular cone the fat has the appearance of large and loosely connected lobules, broken up by the passage of the nerves and vessels. Outside the muscular cone the fat is more firmly packed and contains a quantity of connective tissue, which is closely attached to the muscular sheaths. No doubt this fat and connective tissue serve as a sufficient barrier so that the anesthetic solution is held within the cone, with production of anesthesia or blocking of the nerves that traverse it—the nasociliary nerve, the long and short ciliary nerves, the ciliary ganglion, the optic

nerve, and all the motor cranial nerves except the trochlear, or fourth. The importance of injecting the anesthetic solution into the cone is obvious if complete anesthesia of the globe is to be obtained.

Decrease in intraocular pressure

A decrease in the intraocular pressure occurs after injection within the cone. If the amount of epinephrine hydrochloride (1:1,000) is increased to 3 or more drops to 5 cc. of the anesthetic solution, the decrease in the intraocular pressure is greater, and a decrease of as much as 20 to 30 mm. Hg may be noted in cases of acute glaucoma with high intraocular pressure.

After the injection within the cone, there is often noticeable deepening of the anterior chamber, particularly when the amount of epinephrine hydrochloride is increased. This indicates a decrease in the fluid contents of the eye posterior to the diaphragm. The incision for cataract extraction is thus made easier in cases in which the chamber has been shallow. Also, with lowering of the pressure behind the lens, the anterior capsule seems to become relaxed. In some cases the capsule of the lens, which prior to operation appears to be too tight to be grasped with smooth forceps, is found to be loose enough after an injection within the cone so that a good bite of the capsule can be obtained with the forceps.

With some methods of intracapsular extraction of cataract a decrease in the intraocular pressure seems to be an advantage, as it lessens the possibility of prolapse of the vitreous. This is also true in the removal of intraocular foreign bodies by the scleral route. In extraction with capsulotomy it is often more difficult to express the lens when the pressure is decreased. With deeply set eyes the operation is somewhat facilitated if the eye is slightly proptosed. With lowered intraocular pressure the iris may not prolapse well in corneoscleral trephinations, an incident which may be embarrassing.

The decrease in the intraocular pressure following the injection of procaine and epinephrine within the cone may be accounted for by the constricting action of the epinephrine on the arteries entering the globe, an effect which would tend to decrease the amount of blood entering the globe but would not impede its exit through the venae vorticosae, since the solution does not reach them, located as they are in the episcleral space. The free anastomosis of the venae vorticosae and other orbital veins with the facial vein allows free exit of the blood.

Indications for cone injection

The indications for a cone injection are: globe operations, muscle operations, enucleation, evisceration, and, as suggested by Icaza,[11] in cases of acute glaucoma for the immediate relief of pain. In acute glaucoma the intraocular pressure is also lowered rapidly, particularly if the amount of epinephrine is increased. The pupil is then more easily contracted and the pressure controlled with a miotic. However, Scheie,[12] has found that physostigmine (eserine) has no effect on the pupil for 1 to 3 hours following a cone injection of procaine hydrochloride while pilocarpine promptly constricts the pupil.

Operations on the lacrimal sac

For extirpation of the lacrimal sac quite satisfactory anesthesia may be obtained by infiltration anesthesia around the sac and the injection of anesthetic solution into the sac. However, some pain will usually be experienced when the sac is excised from the nasolacrimal duct, or when the duct is curetted, because the anterior superior alveolar nerve which supplies these parts is not reached. To block the anterior superior alveolar nerve, an injection proximal to where it branches off the infraorbital nerve within the infraorbital canal is indicated because it passes through osseous canals to supply the lower part of the lacrimal sac and the nasal duct and so is not reached by an injection around the sac.

More complete anesthesia for operations on the lacrimal sac may be obtained if the nasociliary nerve is blocked at the anterior

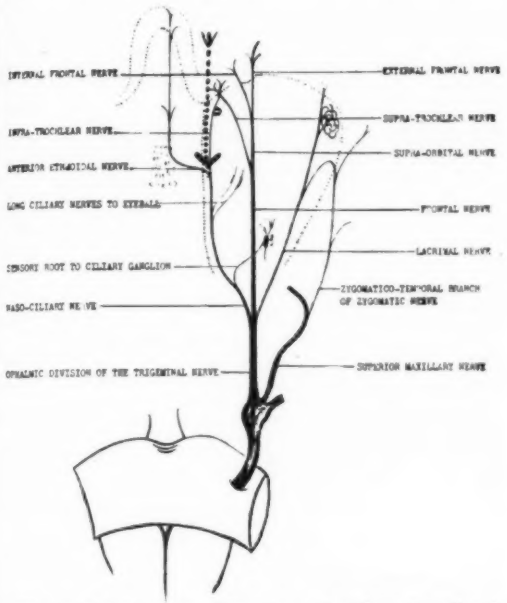

Fig. 8 (Atkinson). Diagram showing the distribution of branches of the ophthalmic division of the trigeminal nerve (after Whitnall). Arrow indicates site of injection for block of nasociliary nerve.

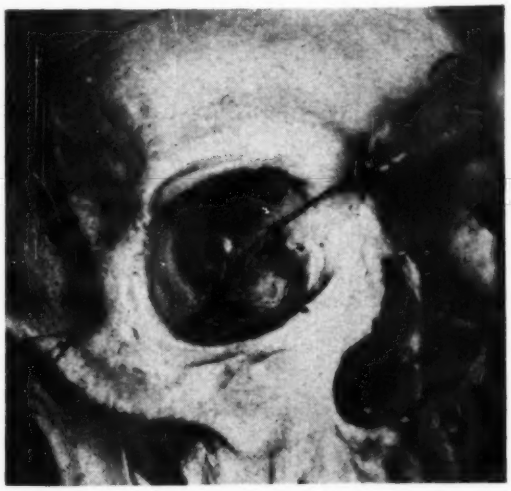

Fig. 9 (Atkinson). Skull showing needle in position to block the nasociliary nerve.

ethmoid foramen so that both the anterior ethmoidal and infratrochlear nerves are blocked (fig. 8).

For this injection, the 3.5-cm. needle is

Fig. 10 (Atkinson). Injection to block the nasociliary nerve.

introduced a little above and to the nasal side of the trochlea (figs. 9, 10). The needle is kept close to the superior nasal wall and at a depth of 3 cm., and 2 or 3 cc. of a 2-percent solution of procaine hydrochloride with epinephrine hydrochloride is injected. On withdrawing the needle another injection is advisable along the orbital margin in the vicinity of the supraorbital notch anterior to the orbital septum but close to the periosteum. Occasionally the angular vein (fig. 11) is pierced, but if the anesthetic solution is not injected into the vein no harm is done,

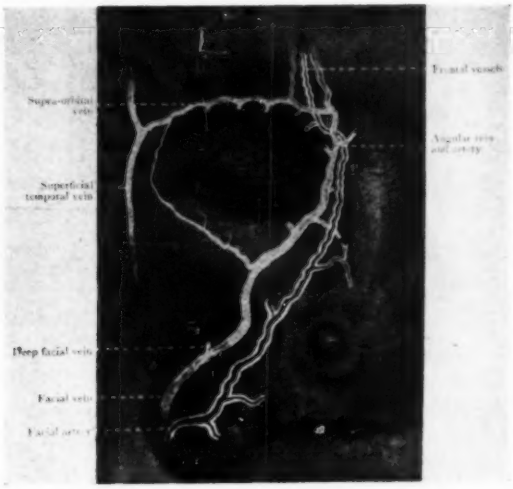

Fig. 11 (Atkinson). Vessels to avoid in block of nasociliary nerve (after Whitnall).

Fig. 12 (Atkinson). Skull showing position of the needle for injection into the infraorbital canal to block the infraorbital and the anterosuperior alveolar nerve.

although the resulting ecchymosis is annoying.

The block of the infraorbital nerve proximal to the anterior superior alveolar nerve may be made in the infraorbital canal or inferior orbital fissure.

To inject the infraorbital canal, the needle

Fig. 13 (Atkinson). Injection into the infraorbital canal.

is introduced 1 cm. from the middle of the ala of the nose and carried almost to the bone. It is then directed upward and temporally at an angle of 20 degrees (figs. 12, 13) and kept in close contact with the bone, injecting the procaine solution as it advances.

The foramen is found more easily if the index finger of the other hand is placed over the foramen, as suggested by Labat,[13] indicating the location of the foramen and the axis of the infraorbital canal. Usually a small depression is felt at the opening of the canal, 0.5 to 1 cm. below the inferior orbital margin, about in line with the supraorbital

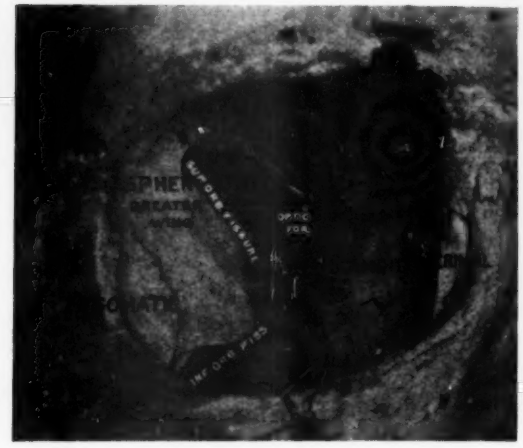

Fig. 14 (Atkinson). Orbit showing inferior orbital fissure and infraorbital canal (after Whitnall).

notch which helps to locate it. The needle is introduced along the canal 0.5 cm. to 1 cm. so that the injection will be proximal to the branch of the anterior superior alveolar nerve.

If the foramen is not found or is not present, as is occasionally the case, the block can be made in the inferior orbital fissure (fig. 14). To reach the fissure, the 22-gauge, 3.5-cm. needle is introduced as for a cone injection. As soon as the point of the needle passes through the orbital septum it is directed toward the floor of the orbit in the same plane as for the cone injection (fig. 15, 16). The point of the needle is moved along the orbital floor from 1.5 to 2.5 cm. until the fissure is felt; it is then introduced 1 cm., and 1 to 2 cc. of the procaine solution is injected.

Infiltration over the site of the sac decreases bleeding but is not necessary for anesthesia. For the operation of dacryocystorhinostomy, in addition to block of the nasociliary and the infraorbital nerves, topical anesthesia of the nasal mucous membrane should be obtained by the use of a nasal pack.

Fig. 15 (Atkinson). Skull showing position of needle for injection into inferior orbital fissure for block of the infraorbital nerve.

Fig. 16 (Atkinson). Injecting the inferior orbital fissure to block the infraorbital nerve.

Field block for anesthesia of the lids

Since there are so much overlapping and variation in the sensory nerve supply of the lids (fig. 17), it is simpler in operations involving a considerable area of the lids to produce a field block rather than to try to block the individual nerves.

Fig. 17 (Atkinson). Right eye: The sensory nerves are indicated by solid lines (after Whitnall). Left eye: Injections for block of lids indicated by arrows.

Injection, using the 3.5-cm. needle, is made at the center of the orbital margin as shown (left eye) in Figure 17, above or below, the position depending on which lid is to be blocked. The point of the needle should follow the orbital margin closely but should not penetrate the orbital septum. A 2-percent solution of procaine hydrochloride with epinephrine hydrochloride (1:1,000) is injected slowly as the needle advances. In order to inject the anesthetic all along the margin and to infiltrate well the tissue around the nerves that emerge from the orbit, it is necessary to withdraw the needle a little and redirect it several times. The injection should not be made just under the skin, but should be close to the periosteum. An extra 0.5 cc. of the anesthetic solution should be injected deep in the vicinity of the supraorbital notch.

After the anesthetic solution has been injected along one half the superior or the in-

ferior orbital margin, the location depending on which lid is to be blocked, the needle is partially withdrawn; it is then redirected and the solution injected along the other half of the orbital margin. Each injection should be carried beyond the line of the palpebral fissure.

For removal of small tumors of the lids, such as papillomas, infiltration at the site of the tumor is usually sufficient and is the simplest method. It also decreases the bleeding. For chalazion and operations involving the margin of the lid which, owing to its rich nerve supply, is extremely sensitive, instillation anesthesia and infiltration at the site of the chalazion are often not sufficient to make the operation painless. However, instillation anesthesia and an injection of 1 cc. of the anesthetic solution through the conjunctiva of the cul-de-sac or through the skin to reach the same location in the sector of the lid where the chalazion is located, will more frequently produce complete anesthesia.

Conclusion

Most eye operations may be safely performed without pain with local anesthesia.

129 Clinton Street.

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ECZEMATOUS KERATITIS AND ARIBOFLAVINOSIS*

H. J. STERN, M.D.
New York
AND
J. LANDAU, M.D.
Jerusalem, Palestine

Eczematous (phlyctenular or scrofulous) keratitis is most commonly regarded as an allergic condition. It occurs mainly in children with so-called scrofulous diathesis. Enlarged tonsils and adenoid vegetations are very frequently present, and the submandibular glands are often found to be enlarged. The disease is characterized by diffuse infiltration of the conjunctiva and the corneal epithelium, with marked hyperemia, and multiple lymphocytic nodules of varying size on the bulbar conjunctiva or at the limbus which may break down and become infected. The condition is often accompanied by intense pain and blepharospasm. Infiltration of the limbus leads to the development of superficial vessels at the periphery of the cornea, a condition which is called eczematous, scrofulous, or phylyctenular pannus. It is common to find eczematous lesions around the nose and the mouth.

The hypersensitivity of the conjunctival and corneal tissues is supposed to originate from a hidden, usually clinically healed, tuberculous lesion. However, it need not necessarily have been provoked by the tuberculous antigen, but may possibly have been induced by other bacterial toxins (hetero-allergy).

Treatment consists of the administration of yellow oxide of mercury, calomel, dionin or atropine, the local application of heat, general improvement of the diet and living conditions, change of climate, ultraviolet irradiation, or cod-liver oil. However, the treatment, which is very tedious, may prove disappointing in many cases, and frequent relapses are the rule.

In recent years several authors have claimed that riboflavin exerts a beneficial effect on a number of corneal diseases of different etiologies, among them eczematous keratitis.[1, 2] A certain clinical resemblance between eczematous keratitis and the acute manifestations of ariboflavinosis has induced us systematically to examine the efficacy of riboflavin in this disease.

CASE REPORTS

Case 1. M. M., a girl aged 10 years, with typical signs of scrofulous diathesis, had been treated at our clinic for phlyctenular keratitis four times in the course of the past four years. On every occasion treatment had to be continued for many weeks before the condition cleared up.

This time she had been under treatment for four weeks (atropine, yellow oxide of mercury, sulphathiazole, and cod-liver oil) when it was decided to attempt riboflavin therapy.

At this stage the patient's eyes were very bloodshot, multiple phlyctens were present around the limbus of both eyes, and the corneal periphery was markedly vascularized. Eczema was observed around the nose and the mouth. The urine contained 250 micrograms of riboflavin per 1,000 cc.

Local treatment was discontinued, and the child received 5 mg. of riboflavin daily. She improved steadily and, after two weeks, the eyes cleared up completely and the facial eczema also improved. The child has remained free from ocular symptoms since that time.

Case 2. A. H., a boy, aged 9 years, had been under treatment at our clinic for eczematous keratitis on four different occasions during the past two years. He was suffering from enlarged adenoids. The ocular condition had always proved very difficult to treat. Superficial and deep vessels appeared in the cornea, and vision was considerably reduced.

When he was first seen this year, both eyes presented the familiar syndrome with blepharospasm, lacrimation, and pain. The bulbar conjunctiva was deeply injected, and the entire corneal circumference was extensively vascularized and showed multiple phlyctens.

The patient received 3 doses of 5 mg. of riboflavin without any local treatment. Within 24 hours both eyes became white and quiescent, and the subjective symptoms entirely disappeared. The patient has not been seen since at our clinic.

Case 3. D. M., a girl, aged 6 years, well developed

* From the Eye Department, Hadassah University Hospital, Jerusalem. Read before the New York Society for Clinical Ophthalmology, May 3, 1948.

Fig. 1 (Stern and Landau). *Case 3.* Appearance of patient after a severe attack of eczematous keratitis lasting five days.

but somewhat puffed up, had been under our care for several years and suffered from frequent recurrences of eczematous keratitis. She reported to hospital after a very severe attack lasting five days. Very marked blepharospasm, lacrimation, and pain were observed; both eyes were bloodshot, and multiple limbal phlyctens, infiltration of the epithelium of the corneal periphery, and corneal vascularization were noted (fig. 1).

The patient received 5 mg. of riboflavin by mouth twice daily without any local treatment. The following day the condition was unchanged. Treatment with riboflavin, alone, was continued. By the fourth day the right eye had become white, the cornea was clear, and the infiltration around the limbus was entirely gone. The left eye, however, was still injected. By the fifth day both eyes were cured (fig. 2). No pathologic changes were found in the nasopharynx.

Fig. 2 (Stern and Landau). *Case 3.* Appearance of the same patient as in Figure 1 on the fifth day after riboflavin therapy was instituted.

Case 4. R. A., a young woman, aged 23 years, was suffering from intense pain and blepharospasm in the left eye, which showed eczematous keratoconjunctivitis with a large single phlycten at the limbus (fig. 3). The corneal periphery of the left eye was vascularized. The right eye appeared normal. The nose was very narrow, and examination revealed a deviation of the septum toward the left and compression of the left lower turbinate.

The patient had received the usual treatment for the ocular condition elsewhere, without any benefit. Local treatment was discontinued and she received 5 mg. riboflavin by mouth twice daily. After two days the eye was much improved; by the fifth day she was completely cured (fig. 4).

Case 5. A. R., a girl, aged 13 years, showing no scrofulous stigmas, applied to the hospital suffering from acute Koch-Weeks conjunctivitis. She

Fig. 3 (Stern and Landau). *Case 4.* Eczematous keratoconjunctivitis with a large single phlycten at the limbus near the 5-o'clock position.

received 2 gm. of sulfadiazine by mouth during one day. The next day the cornea showed multiple phlyctens and vascularization around the limbus in both eyes. The urine contained 260 micrograms of riboflavin per 1,000 cc. Five mg. of riboflavin by mouth twice daily were added to the sulfadiazine therapy. Two days later the eyes had become white and the intense vascular engorgement had entirely disappeared.

Case 6. T. R., a girl, aged 8 years, well developed but undernourished, sister of the patient in Case 5, suffered from acute Koch-Weeks conjunctivitis. Multiple phlyctens were present in both eyes and the corneal periphery was vascularized (fig. 5). She received 2 gm. of sulfadiazine daily by mouth which cured the acute conjunctivitis entirely within two days, but the eyes remained painful and injected. The picture was now that of a primary eczematous keratoconjunctivitis. The urine contained 255 micrograms of riboflavin per 1,000 cc. The patient received 5 mg. of riboflavin by mouth twice daily and within another 48 hours she was completely cured (fig. 6).

Case 7. A. B., a boy, aged 3½ years, pale and

with enlarged submandibular glands, suffered from pain and lacrimation in both eyes. There was some photophobia. Around the limbus of both eyes 3 or 4 small phlyctens were visible, and the eyes were mildly injected. He received 5 mg. of riboflavin by mouth twice daily. Four days later both eyes were white and clear and the phlyctens had disappeared without a trace.

Case 8. R. M., a girl, aged 13 years, exhibited pellagroid pigmentation of the arms, neck, and ears. She had a deeply furrowed, red tongue. Both eyes were very bloodshot, and the patient suffered from intense blepharospasm and lacrimation. Several phlyctens were seen along the limbus in both eyes and the corneal periphery was vascularized. The patient received 5 mg. of riboflavin by mouth twice daily, and the ocular condition had cleared up without local treatment within four days. Fifty mg. of

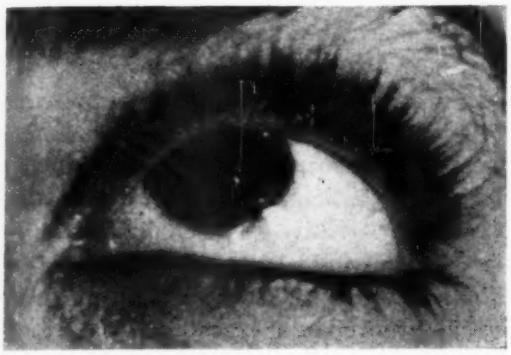

Fig. 4 (Stern and Landau). *Case 4.* Appearance of the same eye as in Figure 3 on the fifth day after institution of riboflavin therapy.

nicotinamide was then administered by mouth twice daily, and the dermatitis and glossitis improved steadily. After one week she was cured.

Discussion

The striking effect of riboflavin in a condition known occasionally to be very refractory to the usual local treatment may be explained in either of two ways: (1) The action of riboflavin is a purely pharmacodynamic one, or (2) eczematous keratitis is a manifestation of ariboflavinosis and riboflavin specifically relieves this deficiency.

Bessey and Wolbach[3] were the first authors to describe in detail the occurrence of corneal vascularization with infiltration of the corneal stroma and epithelium in riboflavin-deficient rats. They regarded this condition as a compensating process, whereby blood is brought into closer contact with the deeper

Fig. 5 (Stern and Landau). *Case 6.* Multiple phlyctens were present in both eyes and the corneal periphery was vascularized.

layers of a cornea deprived of riboflavin.

Riboflavin constitutes the prosthetic group of the yellow respiratory enzyme on which the corneal cells depend for their respiration. This subtle process can be assumed to be disturbed in any pathologic condition involving the deeper layers of the epithelium and stroma.

Such disturbances may be due to the action of bacterial toxins, of chemical substances released by foreign bodies, and also of noxious metabolic products released in allergic reactions (histamine or histamine-like substances).

It is conceivable that in such conditions the administration of riboflavin relieves the disturbance in the respiration of the corneal cells. It has actually been claimed that riboflavin is very beneficial in a number of corneal diseases such as rosacea keratitis,[4-8] syphilitic parenchymatous keratitis,[1, 9, 10] and trachomatous pannus.[5, 11] Riboflavin has further been shown to have a certain antiallergic action.[12, 13] It is therefore possible that the relief of eczematous keratitis by riboflavin is due either to a nonspecific effect on the respiration of the corneal tissue or to the

Fig. 6 (Stern and Landau). *Case 6.* Appearance of the same eyes as in Figure 5 within 48 hours after riboflavin therapy was begun.

antihistamine action of the vitamin in a disease with undeniably allergic characteristics. However, several points argue against this assumption.

The reports on the beneficial effect of riboflavin in corneal diseases other than eczematous keratitis are open to serious criticism. Its curative effect in rosacea keratitis has been denied by a number of authors.[14, 15, 18] Its effect in syphilitic parenchymatous keratitis seems improbable at first sight, and the correctness of the diagnosis has been challenged in some of the cases published.[5, 7] In trachomatous pannus it has been shown by Landau and Stern[11] that the good results obtained with riboflavin may be explained by the concurrent presence of a true ariboflavinosis.

It is a common experience that eczematous or phlyctenular keratitis occurs almost exclusively in undernourished, scrofulous individuals, or in those on a faulty diet, and it is possible that the administration of riboflavin in these cases relieves a subclinical deficiency. Thus the curative effect would have to be ascribed not to a purely pharmacodynamic action but to a kind of adjuvant treatment. Assuming that this is possible, let us consider more closely the assumption that eczematous keratitis is a manifestation of riboflavin deficiency, particularly in view of the abnormally low riboflavin values in the urine of the three cases in which the urine was examined.

A remarkable clinical similarity exists between eczematous keratitis associated with eczematous pannus and the ocular signs of ariboflavinosis. The corneal vascularization seems to be identical with that in eczematous pannus. The cheilitis and the eczema of the nasolabial fold in ariboflavinosis are strongly reminiscent of the facial lesions in eczematous keratitis, with certain differences probably attributable to differences in age of the respective patients.

Mann[16] and Stern[17] have published descriptions of cases of acute ocular manifestation of ariboflavinosis which are very similar to typical cases of eczematous keratitis. The condition involves acute injection of the bulbar conjunctiva with marked vascularization of the cornea, accompanied by edema of the perilimbal conjunctiva and the peripheral corneal epithelium, which may break down and develop small ulcers. It is very painful and causes intense reflex blepharospasm. It seems to occur mainly in adults but eczematous keratitis may constitute a modified picture of the same disturbance in predisposed children. Our reports on the uniformly satisfactory therapeutic results, together with the subnormal urinary riboflavin values in the cases in which the urine was examined seem to justify the conclusion that these diseases are identical.

In many cases eczematous keratitis seems to be an allergic reaction. The patients frequently are scrofulous individuals with inflamed lymphatic glands, and are almost always hypersensitive to tuberculin. In sensitized humans and laboratory animals the condition may be produced experimentally by instilling a drop of allergen into the conjunctival sac (Calmette's reaction). But it is also known that patients of this type frequently have a craving for carbohydrates which they consume in great quantities, a diet likely to lead to ariboflavinosis.

While we are unable to exclude the possibilty of a pharmacodynamic effect of riboflavin in exzematous keratitis, we are inclined in these cases to ascribe the dominant rôle to ariboflavinosis. Thus it may be assumed that a chronic riboflavin deficiency becomes manifest as an ocular lesion of the above-mentioned type in predisposed individuals, mostly children, but sometimes also in adults.

There seems to be a constitutional element in such a predisposition identical with that defined clinically as the scrofulous habitus. These patients usually show the typical changes in the nasopharynx. It is not quite certain whether these represent concomitant (and coördinated) symptoms, or whether the nasal lesions are prerequisite to the ocular

manifestations. The observations of Feigenbaum[20] in cases of unilateral trachoma (or unilaterally more severe trachoma) suggest that the second possibility is more probable in our cases as well. While lymphatic changes in the nasopharynx were observed in the majority of our child patients, it was noteworthy that our one adult patient had her only ocular lesion on the same side as the nasal lesion.

Phlyctens seem to occur fairly regularly in the course of Koch-Weeks conjunctivitis. In 1921, Feigenbaum[21] found this condition in at least 8 percent of several thousands of patients suffering from this conjunctivitis, and concluded that these complications probably represent the reaction of the scrofulous individual to the toxic irritation by the agent. In a personal communication this author has stated that he repeatedly observed similar phlyctens in scrofulous children during a diplobacillary infection, as well as exactly at the point of contact after irritation of the bulbar conjunctiva by an incorrectly implanted eyelash.

It seems, therefore, that it takes two or more factors to produce the clinical picture of eczematous or phlyctenular keratoconjunctivitis, and that one of these is ariboflavinosis. On the other hand, the fact that many cases of ariboflavinosis never display the acute symptoms of eczematous keratitis seems to suggest that a second factor, in most cases an allergic reaction, is necessary.

In view of the effect of riboflavin in eczematous keratitis, further experimental and clinical investigation will be required to elucidate the question whether this condition is due to a nonspecific pharmacodynamic action of the vitamin, or to the specific relief of a riboflavin deficiency in predisposed individuals. A number of facts seem to point to the second possibility, namely that ariboflavinosis constitutes a decisive etiologic factor.

Summary

1. Riboflavin has a marked therapeutic effect on eczematous keratitis.

2. The question whether this effect is due to a nonspecific pharmacodynamic action of the vitamin or to the specific relief of a deficiency is discussed.

3. It is concluded that a number of factors (nasopharyngeal lesions, infectious agents, or mechanical irritation) in individuals with a constitutional scrofulous disposition may release the pathogenic mechanism producing eczematous keratitis, but that ariboflavinosis is the essential basic condition.

600 West 111th Street (25).

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NOTES, CASES, INSTRUMENTS

SIMPLIFIED EXTERNAL EYE PHOTOGRAPHY*

PHILIP H. LANDERS, M.D.†
Baltimore, Maryland

Photography of the external eye is practical with minor modifications of an Argus model C-3. This is a medium-grade, 35-mm. camera with synchronized flash-bulb attachment. On reviewing the description of the mechanisms devised by Bedell,[1] Bogart,[2] Irvine and Stimson,[3] Katzin,[4] and Knighton,[5] it is evident that the stress has been on securing perfect focus and on being able to maintain that focus during the time necessary to manipulate the camera and lighting equipment.

In this camera, the emphasis is on creating a system having greater depth of focus, accuracy of aim, and speed of operation. Minor movements of the patient are not important. It is therefore possible to discard the table, head-holding frame, camera stand, complicated electrical circuits, and interchangeable mounts for the focusing ground glass and camera. The elimination of this complex, costly, and cumbersome equipment frees the camera for use in the clinic, on the wards, and in the operating room. These improvements are obtained by a combined fixed aiming and focusing device and a light of sufficient intensity to reduce the lens aperture to a minimum, thereby increasing the depth of focus.

In the construction of the optical system, parts made by the original manufacturer were preferred. This system consists of the original camera, which is set at 5-foot focus, and a combination of portrait lens chamber and sunshade, which screw to the lens mount. The portrait lens chamber contains a plus-3 lens and a disk of Wratten No.-2 gelatin filter. A plus-16 lens ground from a cataract lens blank, is mounted inside the front of the sunshade. This combination of camera and lenses has a focus of 48 mm., which was determied by the ground-glass method. Chromatic aberrations need not be considered. Spherical aberration is minimized by using a large diameter lens for the plus 16, so that only the central portion is used. The residual aberration is negligible.

Fig. 1 (Landers). Argus C-3 camera with modifications.

A curved metal rod is bolted to the sunshade in such a manner that the tip of the rod is exactly 12 mm. below the center of the photographic field, and is exactly at the 48-mm. distance from the front lens. The size of the object and of the picture is identical. The tip of the rod is at the margin of the field but does not show in the picture.

The light source is a No.-5 press flash bulb, and the film is indoor Kodachrome (type A). The light temperature of the press flash bulb No. 5 is not perfectly adjusted to type-A Kodachrome, but this is corrected by the filter mentioned above.

* Published with permission of the chief medical director, Department of Medicine and Surgery, Veterans Administration, who assumes no responsibility for the opinions expressed or conclusions drawn by the author.

† Resident in ophthalmology, Veterans Administration, Fort Howard, Maryland.

The standard reflector for the camera cannot be used because of the size of its reflection on the cornea. A small metal shield is therefore substituted. This shield is painted black on the inside except for the area immediately behind the bulb. This is to minimize further the size of the corneal reflection. The front of the reflector has a transparent plastic cover to prevent possible injury to the patient from an exploding bulb.

With the press No. 5 bulb only 19 cm. from the eye, the light intensity permits the lens aperture to be decreased to f-16 or smaller. This factor is important because the depth of focus of the system is over 6 mm. at f-18, and only 1 mm. at f-3.5.

The speed of the shutter is not effective until it is faster than 1/100 second because of the few thousandths of a second during which the flash lasts. If it is desirable to reduce the light more than is accomplished by an f-18 opening, the shutter speed can be set faster.

Fig. 2 (Landers). Demonstration of operation of camera.

In actual practice, this camera can be used without loss of more than a few moments, or can be operated by a nurse or secretary. The mode of operation is most uncomplicated. The camera is kept loaded and with the proper adjustment set, with the exception of the aperture which is adjusted to the complexion color. The patient is warned not to be frightened at the flash, and the camera is held before his face in such a manner that the tip of the focusing rod is 12 mm. below the center of the desired field and in the proper plane. The picture is then taken instantly, and a notation made.

Fig. 3 (Landers). Illustration of a normal eye showing size of field and depth of focus.

The same camera as described, with an additional portrait chamber attachment using a plus-3 lens, is excellent for photographing both eyes in cases of squint, or anything requiring a full face. This is accomplished by using 23- or 32-cm. distances with approximately the same settings as to shutter speed and aperture.

In conclusion, it would seem to be of more than casual importance that, with the present trend toward more and more complex, elaborate, and expensive mechanisms, a simple, effective, and inexpensive device can be constructed for making permanent color records of external and anterior eye conditions.

1405 Winston Road (12).

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OCULAR MANIFESTATIONS IN COCCIDIOIDOMYCOSIS

JESSE M. LEVITT, M.D.
Brooklyn, New York

Coccidioidomycosis[3] is a disease caused by a fungus, Coccidioides immitis. It is considered to be primarily a disease of rodents, transmitted to man when dusts that contain spores are inhaled. It is endemic in the San Joaquin Valley in California and in other southwestern states, and has been seen in other parts of the United States. During the war the condition became important because several thousand soldiers, who were stationed in Army camps in the Southwest, probably became infected. Some of these men will undoubtedly develop active pulmonary lesions within the next 10 years.

The lungs are usually first infected through the inhalation of the spores and the infection is spread to various parts of the body by continuity and through lymphatic and vascular channels. Up to the time of World War II, only a severe general form of the disease, resembling tuberculosis, had been recognized. It is now known that in most instances the infection is benign, simulating influenza or virus pneumonia, and lasts usually 3 to 6 weeks. X-ray studies of the lungs in the mild form resemble those shown in virus pneumonia, while in the chronic form they resemble those in tuberculosis, with cavitation a prominent feature.

The causative organism may be recovered from the sputum and other infected body fluids and tissues. Skin tests with Coccidioides-immitis antigen may be helpful in diagnosis. A positive skin test does not necessarily indicate active coccidioidal infection. A negative test is believed to exclude the infection, except where the infecton is too early for sensitivity to have developed. The pathologic condition is essentially a granuloma greatly resembling the lesions of tuberculosis.

The observation of changes in the ocular fundi in a case of coccidioidomycosis is worthy of record. To my knowledge, no previous report has appeared in the literature. Because of the protean nature of the disease and its great similarity to tuberculosis and the other granulomatous diseases, one would expect some cases to have an involvement of the uveal tract and retina.

Charles E. Smith[1] stated that the patient with coccidioidomycosis may exhibit typical phlyctenular conjunctivitis. Willett and Weiss[2] reported the occurrence of conjunctivitis in 4 of 100 cases. With the exception of conjunctivitis, ocular involvement in the course of the disease has not been mentioned in the literature.

REPORT OF A CASE

Following is the report of the eye findings in a case of coccidioidomycosis of the primary pulmonary type. The diagnosis was established by the isolation of the causative agent, Coccidioides immitis, in the sputum. Striking changes were observed in the vitreous chambers and fundi.

History. Sergeant D. T. L., white, aged 20 years, a patient in the medical department of Fletcher General Hospital, Cambridge, Ohio, was referred to me by Dr. Frank J. Mellencamp on August 14, 1943, because of visual disturbances of both eyes.

While overseas he became ill with what was initially diagnosed as lymphocytic choriomeningitis. He was hospitalized in the

British 40th General Hospital in British West Africa and was subsequently evacuated to the United States.

The only complaint on admission to Fletcher General Hospital was pain in the back and legs. Physical examination, including the chest, was negative. X-ray studies of the chest revealed an infiltrate in the right middle lobe which had the appearance of a tubercular lesion. A fungus, presenting typical structure and cultural characteristics of Coccidioides immitis, was recovered from the sputum.

The patient complained that vision of the left eye first became blurred for intervals of ½ to 1 hour and that later similar episodes were experienced in the right eye. For two weeks prior to my examination he had noticed "shimmering spots" first in the right eye and then in the left eye.

Eye examination revealed vision to be: O.D., 20/30, J1; O.S., 20/20, J1; correctable to 20/15, O.U., with a compound hyperopic astigmatic lens of moderate degree; no significant muscle imbalance; small discrete follicles in the palpebral conjunctivas of both lower lids; white globes; small corneas; pupils that reacted actively to light and accommodation.

The pupils were widely dilated with 4-percent solution of homatropine hydrobromide. The anterior segments of the globes were entirely normal on slitlamp study. Both vitreous chambers were clouded by whitish-appearing, globular opacities which, while movable, maintained a relatively fixed position, indicating a basically normal vitreous structure. The optic nerveheads were of good color with slightly indistinct margins due to the astigmatic refractive errors. The nerveheads were not elevated.

In the right fundus there were several small oval-shaped masses of what appeared to be whitish connective tissue, attached to the retina and moving up and down in the vitreous The bases of these masses at the level of the retina were hemorrhagic. These were mostly evident in the nasal and lower fundus about 1½ disc diameters from the margin of the optic disc. Just below the optic disc, there was a denser strand of similar tissue projecting into the vitreous. A like picture was observed in the left eye with a lesion also present below the left disc. There was no abnormality of the retinal blood vessels. The macular zones were free of pathologic manifestation.

The diagnoses were: (1) follicular conjunctivitis, benign; (2) vitreous opacities and retinitis of proliferative type, considered evidence of probable localization of the causative agent in the eyes; (3) compound hyperopic astigmatism, bilateral.

On the day following my examination the patient left the hospital on a 30-day sick furlough. On his return on September 16, 1943, I reëxamined his eyes and found the vitreous chambers entirely clear and the fundi perfectly normal. There was no trace of the previous pathologic findings.

The lesions noted in the retinas in this case, and their rapid disappearance, offer a striking parallel to the type of granulomatous lesions noted in other parts of the body, and the frequent rapid clearing of the physical signs and pulmonary changes visible to X ray.

Summary

The main features of coccidioidomycosis are presented.

Changes in the vitreous chambers and ocular fundi in a case of coccidioidomycosis are described.

Because of the protean nature of the disease and its great similarity to tuberculosis, one would expect some cases to have intraocular involvement. It is suggested that the ocular fundi should be watched during the course of the disease.

It would not be amiss to include a coccidioidin antigen skin test in the etiologic survey of cases of endogenous ocular infection, particularly in veterans of World War II.

991 Ocean Avenue (26).

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PERIPHERAL IRIDECTOMY IN CATARACT SURGERY

A. C. HILDING, M.D.
Duluth, Minnesota

In cataract extraction, I have observed a practice in the use of peripheral iridectomy which does not seem to be quite rational. A peripheral iridectomy is made in the 12-o'clock meridian when there is also a single corneoscleral suture in the same meridian. The reason given for making the iridectomy is to furnish a communication between the posterior and anterior chambers. This is necessary, it is explained, because it is the aqueous in the posterior chamber which causes iris prolapse, not that in the anterior chamber.

This fact has been established for a long time. However, there is another fact which seems to have escaped notice very largely; namely, that, at the time of iris prolapse, there is no anterior chamber. If there were then the pupil could serve better as a communication between the anterior and posterior chamber than a small peripheral hole. The communication must be between the posterior chamber and the out-of-doors. With the escape of its aqueous the anterior chamber has become merely a potential space, and the iris lies in such close contact with the cornea as to form virtually a water-tight seal. It is under these conditions that that portion of the iris which overlies the breach in the incision begins to herniate as the aqueous in the posterior chamber makes pressure. Only one of two things will stop it: either cessation of pressure or escape of aqueous through a hole in the iris which coincides with the breach in the incision. If the iridectomy does not coincide with the breach in the incision, the aqueous will not escape, and prolapse takes place. Therefore, iridectomies at other points, even though fairly close, will not prevent the prolapse.

The incision, when it gives way, usually does so in the middle of the longest unsupported arc. It would not give way at the site of a single corneoscleral suture in the 12-o'clock meridian. Therefore, a peripheral iridectomy in this meridian would seem to be useless. If the incision gives way, it would do so somewhere between this central suture and one or the other of the angles of the wound. If peripheral iridectomies are to be used with a single suture in the 12-o'clock position, there should be two of them, and they should be placed respectively at about the 10:30- and 1:30-o'clock positions. If a single peripheral iridectomy is made at the 12-o'clock position, then there should be at least one suture on each side midway between the iridectomy and the extremities of the incision.

Either of these procedures would give at least three points of protection along the arc of the incision. according to some previous studies,* 4 or 5 points of protection are much better than 3. They may be furnished by 2, 3, or 4 corneoscleral sutures, combined respectively with 3, 2, or 1 peripheral iridectomies. Any one of these combinations gives reasonable protection against prolapse, but the iridectomies should be

* Hilding, A. C.: Experimental and clinical studies on certain safety factors in closure of cataract incisions. Am. J. Ophth., 28:871-885 (Aug.) 1945.

placed between the sutures and not under them. The sutures can all be placed before the anterior chamber is opened.

626 Medical Arts Building.

STREPTOMYCIN IN OPHTHALMOMILIARY TUBERCULOSIS*

PETER SYKOWSKI, M.D., AND WALTER LAWRENCE, M.D.†
Schenectady, New York

Recently McDermott and others‡ reported their results in the employment of streptomycin in the treatment of tuberculosis in human beings. We wish to present a case of ophthalmomiliary tuberculosis treated with streptomycin.

REPORT OF CASE

History. R. Q., a 28-year-old white woman, married, was well until May, 1947, when pharyngolaryngeal symptoms became apparent. These were characterized by dysphagia, hoarseness, and pharyngodynia. For several months prior to this, increasing fatigability was observed. Until July, 1947, her laryngopharyngeal symptoms persisted. Then, an otolaryngologist was consulted and a diagnosis of laryngophthisis was made. Pulmonary X-ray studies revealed an acute miliary tuberculosis. She was admitted as a patient to the Glenridge Sanatorium on August 9, 1947.

Physical Examination. The patient was poorly developed, poorly nourished, and thin, appearing acutely and chronically ill. There was a moderate amount of pallor of the mucous membranes. Only the arytenoid area of the larynx was visible. It was the site of marked edema and of severe injection; on its anterior surface a thin, grayish, diffuse exudation was present. There were no abnormal signs in the lungs.

Laboratory Data. Pulmonary skiagrams showed a fine, disseminated miliary type of infiltration scattered evenly throughout both lungs.

Ophthalmic Examination. Biomicroscopy and ophthalmoscopy revealed negative findings in the cornea, aqueous, iris, lens, and vitreous. The fundus of the right eye showed 5 small, pinpoint-sized, gray-white nodules. Three of these nodules were 2½ disc diameters from the papilla at the 1-o'clock meridian; 2 were at the 6-o'clock meridian. There were no pigmentary changes in the areas adjacent to the nodules.

Treatment and Course. Two days following admission, streptomycin therapy was instituted, 1½ gm. daily in 6 divided doses. The laryngeal symptoms showed an almost immediate response, and the general response was unusually satisfactory. Comparative pulmonary X-ray studies, one month after admission, showed less density, giving the impression of beginning resolution. During the second month after admission, no definite change occurred. By the third month, however, there was definite resolution, with marked clearing of the infiltration throughout both pulmonary fields—the most striking change in the entire serial studies.

At this time, an ophthalmoscopic examination showed that the choroidal tubercles situated at the 1-o'clock meridian had completely disappeared and those at the 6-o'clock meridian previously easily detectable were scarcely perceptible.

SUMMARY

This report of a case of ophthalmomiliary tuberculosis, treated with streptomycin, shows that the use of streptomycin definitely altered the course of the miliary choroidal tubercles and sponsors the conclusion that streptomycin therapy is of value in arresting miliary choroidal tuberculosis.

1330 Union Street.

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*From the Glenridge Sanatorium for Tuberculosis, Dr. James Blake, chief tuberculologist.

†Assistant tuberculologist at the Glenridge Sanatorium.

‡McDermott *et al.:* Streptomycin in the treatment of tuberculosis in humans. Ann. Int. Med., 27:769 (Nov.) 1947.

SOCIETY PROCEEDINGS

Edited by DONALD J. LYLE, M.D.

COLLEGE OF PHYSICIANS OF PHILADELPHIA

SECTION ON OPHTHALMOLOGY

January 15, 1948

DR. PERCE DELONG, *chairman*

OPHTHALMOSCOPY AND OPERATIONS FOR HIGH BLOOD PRESSURE

DR. ARTHUR J. BEDELL (guest speaker) of Albany, New York, said that the majority of hypertensive patients are free from symptoms, and the high pressure is discovered on routine physical examination.

The ophthalmologist often makes the original diagnosis of hypertension and inaugurates the investigations which may lead to an operation. The prime symptoms for which he is consulted are intense, persistent, treatment-resisting, generalized headache, or nuchal, occipital, or frontal distress, frequently most intense in the early morning hours.

Because of visual defects a patient may visit the ophthalmologist who recognizes the causative significance of the vessel changes, the congestion of the fundus, the lack of retinal definition, the retinal hemorrhages, the retinopathy or the papilledema.

Ophthalmologists should be able to state with reasonable certainty that a given fundus pattern indicates a favorable postoperative outcome, one of doubtful value, or a disappointment. They must know the life history of the retinal variations, and estimate the life expectancy of the individual patient. They should render a concise but genuinely explanatory report, including an opinion based on a knowledge of the favorable and unfavorable results following operation, and upon an understanding of the fundus signs.

If this is done, more cases with a good prognosis will be operated upon, and fewer of the hopeless subjected to the trying ordeals from which little or no benefit can be expected. The prognosis is based on the state of the retinal vessels.

The ophthalmoscopic signs which determine the feasibility of operation are correlated. Many exceptions may be made to them and occasionally what seems to be a hopeless case may, by operation, be given a few more years of life.

It must be remembered that hypertension is a complex manifestation of many factors all of which must be evaluated before an accurate prognosis can be given. All advice is predicated on the integrity of the kidneys, heart, and cerebral vessels.

From the ophthalmoscopic standpoint, there are two types of hypertension: one in which there are very slight vessel changes even after many years of high pressure. Eventually, the majority of such patients die from a cerebral or cardiac accident without ever having a retinopathy.

The other, labelled malignant, should be called fulminating because of the rapidly progressing retinopathy. Frequently at the time of the initial examination there are extensive exudates and hemorrhages with or without retinal edema and an irregular outline of the blood column, a spasm, or white-walled sclerotic vessels.

The fulminating type is comparatively seldom engrafted upon the simple form of essential hypertension. There is, however, no definite duration of the benign stage and no reliable guide by which we can prognosticate the imminence of the signs which almost invariably foretell an early death. It is to these potentially fulminating cases that the surgeon's attention is often directed, for without help, such patients are doomed. As a last resort, an operation may be tried; for medical treatment is ineffective.

The severe cases of retinopathy are usu-

ally associated with signs of marked kidney dysfunction, so that they rarely reach the standard of kidney efficiency demanded by the most careful surgeons.

Patients with papilledema and extensive neuropapillary edema should not be subjected to operation.

Discussion. DR. W. EMORY BURNETT: It has been a great pleasure to hear Dr. Bedell, and to consider certain additional helps in choosing patents for sympathectomy for hypertension. As a surgeon, I am a little out of my depth tonight, but can discuss some of the other considerations besides the eyegrounds at least in our selection of these patients.

I think the only thing we have to contribute tonight is the consideration of the patient as a whole, not depending entirely on eyegrounds, as much as we would like to, but feeling that there are multiple other considerations which have to be taken into our estimate. We have been watching very carefully the results of the operations, and comparing them with the forecasts. We have approximately 60 cases now on which we have operated. Some of them are too recent to evaluate properly, and, therefore, it is hard to say what is of great value and what is not.

We started out using the criteria which had been set up for us by the then known authorities in this work, particularly Peet and Smithwick, but none of the items have been too reliable or accurate, and often there is great difficulty even using all of them. Actually the tests on which we have depended most in the past two years have been the production of sympathectomy of a temporary nature, such as tetra-ethyl-ammonium bromide, or chloride, and ascending spinal or ascending caudal anesthesia, which imitate the operation for a few minutes, the latter more accurately than the former, because the tetra-ethyl-ammonium bromide is a total sympathectomy for a few minutes and the spinal is a selective or partial one. So far these have been the best forecasts that we have found. Although eyegrounds have helped tremendously, we have to realize that what is seen is the effect in a local situation, and this does not necessarily mean the entire body is similarly affected.

Another thing that may contribute to the inaccuracy of our correlation between results and forecasts is the fact that, until recently, certain sympathectomies have been rather limited, and limited sympathectomy may not be sufficient to produce the desired effect; thus, the accuracy of forecast is not actually disproved. The eyegrounds and other studies may indicate that a patient is a good prospect, that he is very likely to obtain benefit from sympathectomy. If the operation is too limited to interrupt all nerve connection with the splanchnic plexus that does not actually prove or disprove the point.

Because of this, we began not quite two years ago to do a rather extensive sympathectomy more in the nature of Grimson's type, although he was doing complete lumbar removal as well. We have not done that, but have utilized a transpleural approach so as to get to the entire thoracic chain and easily approach the upper portion of the lumbar chain, and have almost routinely, not quite universally, removed ganglia from thoracic 2 to lumbar 2, inclusive, thus leaving very little of the sympathetic chain still functioning.

The results of this more extensive operation are definitely better immediately and, probably when we have followed these patients long enough to evaluate the late results, we may be able to say that the eyegrounds furnish much more accurate forecasts than we are now prone to believe, probably on the basis that we have not given them a chance to be accurate by doing adequately the job which they indicated we should do.

Results of this more extensive sympathectomy have been much better not only in the percentage of patients who have improved, but in the degree of improvement so that we no longer have to hunt around for 10 mm. of diastolic drop. We are rather disappointed if they do not get 30 mm. or better. There have been some failures in our group. There have been failures despite the in-

clusion of spinal anesthesia and tetra-ethyl-ammonium bromide administration, but they have not been nearly so numerous as previously.

DR. FREDERICK BOTHE: We surgeons have a long way to go to appreciate the finer points of the eyeground study with which you gentlemen are so familiar. However, there are a few things that I wish to discuss from that angle.

You are all familar with the excellent results that Peet has obtained in cases of malignant hypertension. In the early cases I was reluctant to do a sympathectomy once the case was diagnosed as one of malignant hypertension. I did two cases at the request of the patients after I had told them I did not feel sympathectomy would help them. The symptoms, particularly headache, were relieved in both instances, but otherwise they were not benefited. Others have experienced results comparable to mine in malignant cases.

This variation in results could be explained on the grounds that the criteria of eyeground changes denoting the beginning of malignant hypertension are variable in the minds of ophthalmologists.

Sympathectomy was performed in cases of essential hypertension to reduce the vasoconstriction. This constriction is either neurogenic or humoral in origin. In the early sympathectomies only the splanchnic nerves were removed. As the results were studied, some surgeons felt that an improvement could be obtained if a more extensive sympathectomy was performed. Today there are various procedures employed.

Griscom has performed a very radical procedure. He has removed all the thoracic and lumbar ganglia in addition to the splanchnic nerves and the celiac ganglion. Others are not so radical. The procedure Dr. Burnett just described is more radical than that which I use.

At present my routine is composed of excision of the sympathetic cord from thoracic −10 to lumbar −2 ganglion, inclusive, with removal of the splanchic nerves and a piece of the celiac ganglion. We hope that future analyses of the results will decide for us whether the conservative or more radical procedures give the best remote results. The improvement in the eyegrounds seems to be about the same with the various procedures.

In the past few years we have increased the scope of the use of sympathectomy. In the early work it was felt a sympathectomy was not indicated if a patient gave a history of a serious cardiovascular catastrophe. In the light of the excellent results that Peet reported in this type of case, I have tried a few in the past several years. My patients had a history of either a hemiplegia or coronary occlusion. These cases have been followed 2½ to 3 years, and their improvement has justified the procedure in carefully selected cases.

One word as to the return of the hypertension. Let us not forget we are not in any way attempting to cure the hypertension. All we hope to do is to produce a safety valve for these patients in an effort to prevent or postpone the cardiovascular catastrophies characteristic of this disease. Sympathectomy increases capacity of the closed system through which the blood circulates. When the spasm again reduces it to the preoperative capacity the pressure returns, but it is hoped the safety valve persists. Results universally show that many patients have symptomatic relief even though the blood pressure returns to the preoperative level within 3 to 6 months postoperatively.

DR. GLENN G. GIBSON: Dr. Bedell is better equipped than any man in the world to bring this message to us, because of his extensive studies on over 2,000 cases of hypertension, which he has recently reported, and also because of his unexcelled fundus photographic material.

The surgery of hypertension has placed an additional burden on ophthalmologists in their diagnosis and classification of these vascular disturbances. When an ophthalmologist is called in consultation on a patient with hypertension, he is asked to make a prognosis. That prognosis is composed of

two parts. The first part is how much damage has been done to the patient's vascular tree. The criterion for that is how much sclerosis is present.

Dr. Bedell has mentioned that the classification of sclerosis into four main types, as advocated by Wagner, is very difficult to do correctly, and he objects to it on that basis. I agree with his objection that it is difficult to learn. In teaching residents, it is one of the most difficult tasks that we have in the whole field of teaching of ophthalmology. However, once students of ophthalmology do learn this classification, they have a tool which is very useful. At least that has been my experience in the past 15 years.

The thing I was taught that stood up best under clinical trial is the classification of hypertension into four grades of sclerosis as taught to me by Dr. Henry Wagner, who devotes most of his time to the classification of these vascular diseases. He believes thoroughly in this grading of sclerosis of the arterioles, and we have found it to be a very useful thing in the study of these cases.

The second part of the prognosis is what is the future course of this case most likely to be. We feel we can estimate this to a certain extent by dividing hypertensive cases into two main groups. The first of these is a chronic hypertension which takes a very chronic course, and the second is what we refer to as a more active type of hypertension which runs a more fulminating course.

What are the eye findings which differentiate these two different clinical courses? In a case of chronic hypertension, the most important one is arteriovenous compression. We attach more significance to arteriovenous compressions than Dr. Bedell seemed to employ in his cases. We feel that arteriovenous compression is the most reliable sign of chronic sclerosis. The other sign which is most important in the chronic group is the color of the vessel. In early chronic hypertension there is a mild accentuation of the reflex stripe and, as this condition becomes more marked, the change is that of the so-called copper-wire artery. The silver-wire changes represent the more marked degrees of chronic sclerosis.

Turning to the other extreme, what are the changes associated with active hypertensive disease? They are: (1) Marked attenuation; (2) presence of angiospasm and intermittent caliber constrictions; (3) presence of edema localized in the retina to an arterial distribution; and (4) presence of the various types of retinopathy, particularly edema of the discs. These signs are the signs of activity of the more rapid fulminating types of hypertension.

Dr. Bedell has made some very dogmatic statements, and has some pretty set ideas on which cases are suitable for surgery and which are not. We feel that our data are much too incomplete, both as to number and as to duration, to assume any dogmatic point of view about which cases should be surgical and which should not. Therefore, what we have to say in this regard is to be taken as conjectural and not as definite established fact.

Analysis of our data suggests that the prognosis is best in those cases where there is grade-one sclerosis. I am not sure that is a very significant statement, because the prognosis in grade-one sclerosis is better than in the more severe grades of sclerosis. The next trend that we find in our data is that those cases which show signs of activity, mainly the vasospastic group, are the ones which show the most convincing evidence of improvement following surgery. Conversely, the more chronic cases show less encouraging results. It is only fair, however, to point out that these are only trends, because there are examples of successful results in both groups, but by and large the more active group shows the better response.

One of the main difficulties in this problem is to differentiate between the retinopathy of acute vasospastic hypertension and retinopathy of malignant hypertension. Let us be fair about this and admit that the classification of the serious forms of this disease is unsatisfactory.

Dr. Bedell implies he does not like the term malignant hypertension. I am only too willing to agree with him that the classification is unsatisfactory as it stands. I think that one of the important contributions of surgery in this field may be that classification and understanding of hypertension will be on a higher plane than it was in the pre-surgical days.

Early in the history of this condition, we felt that the main benefit from surgery was relief of symptoms, and that, even though the blood pressure dropped temporarily, there was not much in the way of permanent value to be had from this operation. We particularly felt that malignant hypertension was much too serious a disease for surgical approach.

Dr. Burnett has operated on a number of cases of malignant hypertension and, from a review of the records, they seem to show the best results. Dr. Bedell states that these cases should not be operated and, on a logical basis, I would be inclined to agree with him, but some of the cases that have had malignant hypertension have shown a number of different signs and symptoms of improvement. They have received benefits which could not be expected by the ordinary remissions that occur in these cases. The results of Dr. Burnett's surgery have changed my opinion of this problem from one of complete disbelief in the value of the procedure to one of cautious enthusiasm. The postoperative fundus findings are unpredictable, but are dramatic in some cases.

Dr. Arthur J. Bedell (closing): Dr. Burnett, I am deeply grateful to you. You have given us the benefits of your mature deliberations. I was told before I came to Philadelphia that the address of a guest was not discussed, and I asked that the habit be broken. How fortunate that the rule was laid aside. You were gracious to come and tell us about your long experience in the surgical treatment of hypertension.

You, Dr. Burnett, have added much to our knowledge. I have, of course, read the literature, and as a result have been left in a state of confusion. The reports from the various clinics have shown that some surgeons go high in the thoracic region and some low in the lumbar, and all have claimed good results; so I have felt that perhaps it was the skill of the operator rather than the extent of the operation that led to the favorable outcome.

I did not tell ophthalmologists the details of operations for I felt that, if they were sufficiently interested, they would read about them. You, however, have saved them the effort, and I am sure they are grateful to you for the clear exposition of the nature of the present operation.

Dr. Bothe, you have added much to the value of the exposition. You have helped us understand some of the problems you encounter and overcome. You have referred to the Peet summaries. In going over his last report, I believe it is the latest one, he reported 1,500 operations. From an ophthalmic standpoint, and, of course, that is all I am discussing, his citations are rather incomplete. He had done things that I would not wish to have done to me. He has reported some conclusions at variance with other surgeons.

Your statement regarding hemiplegia has been of great interest for your good results are surprising. They cause us to pause, and to be more cautious in excluding this type of case from possible benefits of operation.

Dr. Gibson you have added much to the value of the paper, because you have amplified and clarified many of the points which I have touched upon, and brought into relief some questions in which there is a difference of opinion. Your stand has been made so clear that we all recognize your extensive experience and the value of your deductions.

M. Luther Kauffman,
Clerk.

MEMPHIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

March 9, 1948

Hysterical amblyopia

Dr. James E. Wilson reported that J. F. W., a 36-year-old white man, complained of sudden loss of vision. His wife gave a history that he had always been quite a nervous person. He said that his father at the age of 33 years had suddenly lost his vision while walking down the street. His father's vision never returned. Report on the father from a soldier's home reported optic atrophy, cause undetermined.

For a few days before coming to my office he had said that he had felt a roughness and saw specks before his eyes. On January 6, 1948, his vision was good when he went to bed but when he awoke on January 7, 1948, he could not see.

At interview on this date he denied seeing light but at examination admitted seeing 20/200. The pupils reacted normally. The fundi were normal except for a slight temporal pallor of the papilla and a temporal choroidal crescent. Extraocular movements were normal, as were his eyes in every other respect. A blind person will face the examiner to converse but Mr. W. sat semirigid not turning his head or eyes. Spinal puncture and encephalogram were normal. Field examination could not be done.

His wife was told that he had hysterical amblyopia and that his vision would return as suddenly as it had left him. In spite of warning she told this to the patient so that the planned treatment could not be effectively used.

He was hypnotized and given sodium pentothal anesthesia without establishing his ability to see. Reëxamination on January 30, 1948, showed his eyes and condition to be unchanged. On February 20, 1948, at a special church prayer service Mr. W.'s eyes were anointed with olive oil and his vision suddenly returned. Now he is planning to enter the ministry.

Welfare agencies seemed determined to help this "blind man" but were advised that any help would prolong his disability.

Removal of orbital tumor

Dr. J. Wesley McKinney reported the case of J. S., a girl, aged 11 years, who was first seen in the Pediatrics Department of John Gaston Hospital on April 24, 1945. At that time she had a marked swelling of her right eye. This swelling had increased in the past five days, but the right eye had always been larger than the left. A rash was present over the entire body, there was a marked photophobia, and the patient was having frequent headaches.

Temperature on admittance was 103°F. Physical examination revealed a marked swelling of both upper and lower lids of the right eye. The cornea was not ulcerated and the fundus looked normal. There was a marked swelling of the nasal mucous membranes which were red and congested. A vesicular eruption covered the entire body.

Diagnosis on admittance was (1) chicken pox and (2) orbital abscess or cellulitis possibly from rupture of ethmoid or antrum sinuses.

The patient received penicillin, sulfadiazine, cold compresses to the eye, and supportive treatment. Two days after entrance, an external ethmofrontal drainage and a simple antrotomy were performed. No pus was found in the ethmoids or frontal sinuses and only a small amount of pus was encountered when antrotomy was done and the conjunctiva incised and probed. She improved under treatment and was discharged on the 15th hospital day, reporting to the Ophthalmology Clinic one week later.

At this time the examining doctor stated that the right eye showed marked exophthalmos with a dilated pupil which was fixed to light. The right fundus revealed a small red area in the macula, a bit larger than the

size of a pinhead. Vision was: O.S., 20/20; O.D., gross perception of light only. The exophthalmos, as noted, was extreme. No measurements were made since no exophthalmometer was available in the clinic. A tumor mass could be palpated deep in the orbit.

She was sent back to the hospital for removal of the orbital tumor. Repeated X-ray studies were entirely negative. However, on June 22, 1945, the orbit was entered by the Krönlein method. A wedge-shaped sector of the lateral wall of the orbit was resected and left attached to the periosteum. By finger dissection in the depths of the orbit, a well-encapsulated tumor was separated from the optic nerve. It was found to be attached by a broad lease to the greater wing of the sphenoid. There was rather profuse bleeding on reaching the base of the tumor. A tonsil snare was looped over the tumor to the base which was cut by this instrument. Fortunately, there was no further bleeding. The tumor was apparently a larger hemangioma. The child made an uneventful recovery. Vision is now light perception; ocular movements are normal.

Pencil penetrates orbit

Dr. William F. Klotz presented G.E., aged seven years. The child was seen about one hour after having fallen on her way to school, sticking a pencil into her face. She had a gruesome appearance with one inch of the eraser end of the pencil protruding from the face about the level of the lower orbital margin. X-ray films showed the pencil extending back through the orbit and the point sticking into the body of the sphenoid. It appeared to stop within about 2 mm. of the sella turcica. The child was anesthetized with pentothal and the pencil, stuck tightly, was removed with a rotary motion, leaving no portion in the wound. The wound healed within 2 or 3 days without infection, leaving a very small linear scar. She now has vision of 20/20, normal visual field, and no disturbance of any of the extraocular muscles. The mark on the pencil shows the depth, measuring 6 cm., that the pencil had penetrated. The X-ray films show the pencil in position and the postoperative X-ray picture shows no indication of a foreign body.

Ammonia burn of corneas

Dr. M. B. Seligstein presented a case of ammonia burn of both corneas. While the patient was tearing down a 3-inch ammonia pipe on December 29, 1946, some of the liquid squirted through a leak in the gasket in the values, and hit his face, catching the mouth, eyes, and lids. He was sent to St. Joseph's Hospital by the company physician.

Examination the next day showed that both lids and eyes were inflamed, as was the conjunctiva. The cornea of the right eye showed a large stain involving three fourths of its area. A streak about 1½-mm. wide spread from the center of the left cornea toward the 3-o'clock position. Holocaine and metaphen ointment were applied, together with cold boric compresses and atropine. Daily treatment at the hospital brought marked improvement and the patient was allowed to come to the office for treatment.

On January 4, 1947, the left cornea was clear and showed no staining. The right cornea still stained below the center. Gradual improvement of the right eye was noted until January 15th, when the patient complained of severe pain in the right eye. Tension was 28 mm. Hg(Schiøtz) and there was again well-developed staining of the cornea. He was put on eserine ointment for 24 hours but severe pain continued.

On January 16th, there was a small amount of pus in the anterior chamber of the right eye. Precipitates on Descemet's membrane, tension of 32 mm. Hg, and posterior synechias were present. Instillation of 10-percent neosynephrin was started at once, and in addition atropine ointment (1 percent) was used.

By January 20th, the hypopyon had disappeared, the pupil was semidilated, tension

was 26 mm. Hg, and 10 cc. of sterile milk were injected. The next day, tension was 26 mm. Hg, and the patient was still being treated with neosynephrin, atropine, and dionin. Iritis was still present and the cornea stained below. During the day, the patient was fairly comfortable but had pain at night.

On January 24th, tension returned to normal and he was comfortable, with no pain. Since that date, tension has remained normal. Synechias were present but the pupil was kept partially dilated. There was still cloudiness of the cornea in the area below the deepest burn but it did not stain.

The patient again had severe pain and watering of right eye on March 4th. Examination showed a well-developed staining of the cornea, but tension was normal. Atropine and dionin were used in addition to 10-percent neosynephrin. There is still some staining present below; some corneal opacity is present; the pupil is semidilated; tension is normal; vision is 20/200.

Late fistulous wound with epithelial ingrowth following cataract extraction

Dr. Philip Meriwether Lewis presented O. G. H., a white man, aged 57 years, with an unusual series of complications coming on late after an apparently successful cataract operation.

The patient had noticed his vision failing for about one year. He had been under the care of Dr. J. B. Blue who diagnosed a posterior cortical cataract of both eyes and referred him for operation. Corrected vision was 20/100, J14 in each eye.

On January 30, 1947, a combined intracapsular extraction was performed on the left eye and four days later the same procedure was done on the right eye. No difficulty was encountered and no vitreous was lost. Stallard sutures were used on both eyes. The anterior chamber was slow to become restored in each eye, the left requiring 4 days, and the right, 6 days. Convalescence was otherwise uneventful. On April 1st, he obtained on refraction a vision of 20/20 plus in each eye and with both eyes together 20/15. A +2.50D. sph. addition gave a near vision of J1 with an unusually good range. He obtained his glasses on April 11th, and resumed his work as an accountant.

Twelve weeks after operation, on April 24th, he returned because his left eye had been sore for two days and the vision in it had become blurred. Examination showed that the anterior chamber was entirely flat and vision reduced to 20/40. The eye was slightly congested. No leak could be discovered with fluorescein. Atropine was used and a dressing applied. By the following day the anterior chamber was restored. The next day it was lost again and minute leak was discovered in the 12-o'clock meridian. The area was cauterized with trichloracetic acid and a firm dressing was applied.

For about a week the anterior chamber remained normal but on May 7th it was flat again. The actual cautery was used without benefit. On May 10th, he was readmitted to the Memphis Eye and Ear Hospital and a conjunctival flap was dissected up from the limbus. The limbal wound was cauterized with actual cautery and then the flap drawn over it and fastened with heavy silk sutures. Following this the anterior chamber was restored and has so remained.

After about 10 days, the flap slid off but the eye remained highly congested. Intraocular pressure now rose to 35 mm. Hg, (Schiøtz) and there was considerable iritis. Eserine was used for a short time, but mydriatics were employed most of the time. The tension remained around 25 mm. Hg usually, occasionally rising into the 30s. The iritis continued in spite of the regular use of atropine, injections of foreign protein, and salicylates.

Complete physical and laboratory examinations and also X-ray studies of teeth, sinuses, and so forth were negative. On about August 1st, a whitish-looking membrane appeared in the anterior chamber just beneath

the apex of the wound. This growth enlarged very slowly.

The patient was referred for X-ray treatment and to date between 600R. and 700R. have been given. Apparently the epithelial ingrowth had become inhibited. The eye was less congested and less sensitive.

This case was unusual in that no complication developed until 12 weeks after cataract operation, when a leaking wound with loss of the anterior chamber developed. This was followed by irtis, secondary glaucoma, and finally by an epithelial ingrowth. It was felt that if the X-ray therapy failed to control the condition, further surgery might be attempted.

Berman locator brings success

Dr. Philip Meriweather Lewis reported a case in which the Berman locator proved of great value.

B. N., a colored man, aged 37 years, was struck in the right eye while using a hammer in the repair of some machinery. When seen the following day, there was a 2.5-mm. horizontal wound in the center of the cornea. There was a wound also in the lens, which was milky throughout. Vision was reduced to counting fingers. He was sent to the Memphis Eye and Ear Hospital for X-ray localization.

The report showed the foreign body to be just outside the sclera in the inferior nasal quadrant. The globe was of normal firmness, which suggested that the sclera had not been perforated. Application of the giant magnet to the cornea gave no response. An exposure was made of the sclera in the inferior nasal quadrant.

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Recorder for Eye Section.

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EDITORIAL STAFF

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700 North Michigan Avenue, Chicago 11
WILLIAM H. CRISP, *Consulting Editor*
1276 Emerson Street, Denver 3
LAWRENCE T. POST, *Consulting Editor*
640 South Kingshighway, Saint Louis 10
WILLIAM L. BENEDICT
The Mayo Clinic, Rochester, Minnesota
FREDERICK C. CORDES
384 Post Street, San Francisco 8
SIR STEWART DUKE-ELDER
63 Harley Street, London, W.1
EDWIN B. DUNPHY
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Sherman Oaks, California
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324 East Wisconsin Avenue, Milwaukee 2
PARKER HEATH
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M. URIBE TRONCOSO
500 West End Avenue, New York 24
F. E. WOODRUFF
824 Metropolitan Building, Saint Louis 3
ALAN C. WOODS
Johns Hopkins Hospital, Baltimore 5

KATHERINE FERGUSON CHALKLEY, *Manuscript Editor*
Lake Geneva, Wisconsin

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the apex of the wound. This growth enlarged very slowly.

The patient was referred for X-ray treatment and to date between 600R. and 700R. have been given. Apparently the epithelial ingrowth had become inhibited. The eye was less congested and less sensitive.

This case was unusual in that no complication developed until 12 weeks after cataract operation, when a leaking wound with loss of the anterior chamber developed. This was followed by irtis, secondary glaucoma, and finally by an epithelial ingrowth. It was felt that if the X-ray therapy failed to control the condition, further surgery might be attempted.

Berman locator brings success

Dr. Philip Meriweather Lewis reported a case in which the Berman locator proved of great value.

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gress has never returned to the United States. Many notable figures attended this convocation. The opportunity to meet them constituted one of the most important features of the congress for United States ophthalmologists.

In 1924 was published one of William C. Finnoff's famous papers on "Changes Found in Eyes of Rabbits Following Injection of Living Tubercle Bacilli into the Common Carotid Artery." It was a great loss to ophthalmology that one so gifted must die so young. An accompanying paper "Changes in Eyes of Rabbits Following Injection of Dead Tubercle Bacilli into Common Carotid Artery" appeared later in the year.

In 1926, John Evans published a preliminary report on retinal visual scotoma, the beginning of exact and beautiful studies that he has continued to make through the years. A comprehensive article appeared in July of the same year.

In 1928, M. H. Post made a first report on his studies on the sterilization of instruments. In 1928 also, S. Judd Beach began to write about the selection of test types for refraction. It is interesting to note how consistently ophthalmologists have maintained through the years their interest in and studies on subjects that once attracted them.

William H. Crisp replaced Edward Jackson as editor of the JOURNAL in 1928 and retained this position for three and a half years at which time he planned to be absent from the country for a while and so turned over the editorship to the writer. Dr. Crisp has always been interested in higher culture, having himself a remarkable knowledge of literature and languages with many of which he has conversational ability. The JOURNAL during his editorship gave evidence of his exactness as a student. One of his first articles while editor was on "The Literary Phase of Ophthalmology." Long before this he had popularized Jackson's cross cylinder by demonstrating its use and by writing clarifying articles on the subject.

Of historical interest in Volume 13 is the paper by W. H. Wilmer on "Some of the Early Eye and Ear Infirmaries in the United States and the Men Who Made Them."

At this point it might be interesting to JOURNAL readers to know something of the financial policy and history of the JOURNAL. Dr. Jackson, when he amalgamated the other periodicals with the AMERICAN JOURNAL OF OPHTHALMOLOGY, believed that the publication would prosper and represent the profession best if a large number of ophthalmologists rather than only a few had a financial interest in it. He, therefore, formed a stock company owned entirely by oculists who were encouraged to subscribe to one or more shares up to a limited number. Many eye physicians were interested and purchased stock which sold at $20 a share. This method of underwriting has remained throughout the years and from time to time when a few shares were offered for sale, the JOURNAL has purchased them and resold them to ophthalmologists. Jackson also established a policy of paying small dividends to stock holders and putting any excess profits back into the magazine to make it a better publication. A policy of paying for articles accepted for publication was followed for a few years, but then discontinued. The JOURNAL has been financially successful so that dividends were paid in all but two years which were in the depth of the depression.

When Dr. Crisp relinquished the editorship in 1931, Miss Emma Buss joined the staff as manuscript editor. She served efficiently and devotedly until her death, 15 years later. She gave unstintingly of herself to this work, having a fine mind and a splendid education, and was most helpful in the mechanics of editing.

For two or more decades, the leading figure in the world of ophthalmology was Ernst Fuchs of Vienna. His death, in 1930,

was memorialized in the January, 1931, issue of the JOURNAL by tributes from many of his former friends and students. From about 1870 until the first World War, Vienna was the Mecca of American ophthalmologists. From the early 1880s, Professor Fuchs's clinic was the most popular in the world. After World War I he brought his teachings to America, since Austria was no longer in a position to care for foreign ophthalmologists.

In July of 1931, the writer succeeded Dr. Crisp as editor of the JOURNAL. No radical changes took place since the new editor was in accord with the policies of his predecessors. Two years later, however, the editorial board underwent several changes. Clarence Loeb retired from the staff because of incapacitating illness which not long thereafter claimed his life. His loyal and long-time service with the JOURNAL was deeply missed. Rommel Hildreth, Park Lewis, and M. F. Weyman were added to the board. The death of the last named shortly thereafter robbed the profession of an extremely promising young man who had already been appointed associate professor of ophthalmology at the University of Southern California and departmental head in that institution.

In 1932, Otto Barkan described a procedure for the extraction of soft cataracts since widely associated with his name. In the September and October issues appeared articles on historical and experimental keratoplasty by Ramon Castroviejo which did much to popularize this operation. In the 16 intervening years many other articles on this subject have appeared in the JOURNAL, the technique and results continually improving.

Throughout the years distinguished foreign ophthalmologists have frequently contributed original papers to the JOURNAL. In 1933 alone were included such men as Lieut. Col. R. E. Wright of Madras; Ignacio Barraquere of Barcelona; R. Sondermann of Berlin; Carlos Charlin of Santiago, Chile; and T. Wassenaar of Pretoria, South Africa.

In the first issue for 1934, the leading article was on retinal detachment by Clifford B. Walker. Already well known for stressing the value of tiny test objects in taking fields of brain-tumor cases, he spent his later years in a meticulous study of retinal detachments, and was undoubtedly one of the most important figures in the development of the surgery of this condition. This was the first of many articles published by him in the JOURNAL on this subject.

The year, 1934, saw also a paper by George H. Stine on "A Method for Accurate Localization of Retinal Tears." This is probably the method most frequently followed to this day by ophthalmic surgeons in this operation.

In this volume we also find one of the many papers by that most able laboratory worker, Jonas S. Friedenwald, this one on "Retinal Vascular Dynamics." Throughout the years his alert mind has continued to explore new paths. His Jackson Memorial Lecture this year described his utilization of a new staining method for polysaccharides and mucoids that had been used for general pathology and applied by him to the eye. In his preliminary report it was obvious that a new field was opening in ocular physiology and pathology. His thesis on this subject will be published in a later issue of the JOURNAL.

In 1938 and 1939, a remarkable series of 12 lectures on motor anomalies by A. Bielschowsky appeared in the JOURNAL. These were considered so valuable that they were later published by Dartmouth College as a monograph and have served as a text for many students since then.

The editors have always had a keen interest in research and early made arrangements to publish the proceedings of the Association for Research in Ophthalmology. These papers have been a valuable addition to the JOURNAL and at the same time the JOURNAL's coöperation has been a real help

to the Society. Another effort sponsored by the JOURNAL has been the publication of the papers read at the joint meeting of ophthalmologists and orthoptic technicians in connection with the meeting of the American Academy of Ophthalmology and Otolaryngology each year.

In 1941 the writer, having served 10 years as editor, deemed it wise to pass this editorship on and suggested the name of Derrick Vail to the directors who appointed him editor-in-chief. One cannot praise too highly the efficient manner in which he has carried on since that time. He has been able to obtain the best papers and to increase both the size of the JOURNAL and the list of subscribers. This list is now well above 4,000, including many subscribers in foreign countries.

Before the first World War there were no ophthalmic publications equal to those of the Germans. For a time it appeared that these would be successfully revived, but the events preceding the second World War and the war itself obviously have reduced European ophthalmology to a very bad state. It now devolves, therefore, upon England and the United States to carry the torch for the world until such a time as our European brothers can again light their lamps for the benefit of us all.

Lawrence T. Post.

BOOK REVIEWS

MODERN TRENDS IN OPHTHALMOLOGY. Edited by Arnold Sorsby. 2nd edition. Clothbound, 600 pages, 169 illustrations, 3 color plates. New York and London, Paul B. Hoeber, Inc., Medical Book Department of Harper & Brothers, 1947. Price, $12.50.

This book has provided an important part of the reviewer's technical summer reading. The intention had been to give it a rather brief examination since the first edition of seven years earlier was well known. But so much change has taken place in ophthalmology and general medicine during that period and the book contains such a large amount of this new material that it was found to be too interesting to hurry through.

The book is composed of 48 monographs on ophthalmic subjects of unusual interest. Many authors who have exceptional information about them have contributed the chapters. There are five main divisions: Physiology, Diagnostic Procedures, Pathology, Treatment, and Social Aspects.

Part of the first section will be unfamiliar to many ophthalmologists and the most difficult to read. If they will take the necessary time, however, they will find a rich reward. Even if the ophthalmologist is familiar with the basic principles, he will find much recent experimental work described that has materially changed the current thinking on the topic. Chapters that are particularly intriguing are those on the mucopolysaccharides and mucoids, and the one on variations and correlations of the optical components of the eye. F. P. Fischer has written a provocative chapter on senescence of the eye.

John Evans is always convincing when he writes about angioscotometry; so much so in this chapter that the reader may wonder why he has not spent more time on this technique. Probably the inhibiting factor is the very lack of time. This presentation forms a good antithesis to the following one on ophthalmodynamometry by Espildora-Luque, especially on the theories of glaucoma. Evans considers it possible to make a diagnosis of pregnancy in very early stages by angioscotometry.

It should be noted that each subject in the book is only briefly considered and not at all intended as a complete monograph on the subject. It considers only some point that has interested the author. The reader is in fact in danger of being carried away by the writer's enthusiasm until he reads further and finds, perchance, a contrary view

equally ably advocated by someone else!

A fine example of contrary viewpoints is that of the two adjoining chapters on focal infection, one by Woods and one by Berens; the former thinks it plays a very small part in uveal disease, whereas the latter contends that virulent coliform bacilli located in the throat and nose may be of serious import in many cases.

Ballantyne and Michaelson present a good discussion on "Affections of the Retinal Veins," and Wagener and Rucker on "Eye Changes in Blood Dyscrasias."

Stina Björk in writing on "Hypertensive Retinopathy" points out that many observers cannot concur completely with Bailliart and others in their conclusions regarding the relationship of retinal arterial hypertension and systemic arterial hypertension.

There are good chapters on X-ray and Grenz-ray treatment.

In another section Weekers states that 40-percent alcohol retroörbital injections are not used nearly often enough and overlooked in many conditions in which they are helpful.

All in all we have here a most instructive and entertaining book which will enable the ophthalmologist to keep up to date in his thinking.

Lawrence T. Post.

CHILDREN'S EYE NURSING. By James H. Doggart. London, Henry Kimpton, 1948. 144 pages, 93 illustrations including 13 in color, index. Price, 9 shillings, 6 pence.

This is a useful little manual of eye nursing in general, with particular mention of that pertaining to children. It contains a brief description of the anatomy of the human eye and adnexa and a discussion of its more usual afflictions and their nursing care based on a knowledge by the nurse of what the physician sees and does. This is a sound approach because the more the nurse knows and understands of ophthalmology the better is her professional attention.

One of the most useful chapters is that on instruments and dressings. It is generously illustrated with full-size cuts of the more common eye instruments. A description of their care and storage merits especial attention, for in many places the delicate tools of our trade are often cruelly manhandled.

It is too bad that the title implies that eye nursing for children is different from that for the adult. This is not so, as we all know. The use of the manual would not be quite so restricted either if the title were not. It is a good job and is recommended not only for the ophthalmic nurse but chiefly to those beginning that specialty.

Derrick Vail.

TRANSACTIONS OF THE SOCIÉTÉ FRANÇAISE D'OPHTHALMOLOGIE. Volume 60, 1947.

This volume contains the report of the scientific sessions from May 18, 1947, to May 22, 1947. Sixty-three papers and their discussions are presented.

The demonstration of Professor Bonnet's ophthalmoscopic atlas was one of the most important features in the symposium on fundus diseases. Toulant, Beuhamou, and Foissin report on an epidemic dacryoadenitis, characterized by the severeness of the general symptoms, the mildness of the local disease, a fall in the blood proteins, and a high eosinophylia. H. Pagès and J. Duguet describe brawny ectropion as a rare complication of Koch-Weeks conjunctivitis. Dejean and Gassene used a full-thickness postauricular, skin-hair flap in the reconstruction of a burned upper lid. F. Thièbauts, L. Guillaumats, and P. Bregeats present their study of relative hemianopia, with a new perimetric technique—the simultaneous examination of the nasal and temporal fields in one eye or the corresponding fields in in both eyes.

A. Busacca has a detailed paper on the lymph channels of the normal and diseased

conjunctiva made visible by the injection of Trypan blue. R. Nataf reports his studies on trachoma resumed after the war years.

Primary edema of the cornea was discussed by L. Paufique, H. Chavanne, and J. Prost. They studied, clinically and anatomically, eight cases of this rare and mysterious disease which they suggest should be treated by lamellar keratoplasty.

In the symposium on keratoplasty, an anatomic and clinical observation of a nonperforating graft is recorded by L. Paufique and Guy Offret. The graft survived.

Jean Sédan and G. Farnarier discuss the spontaneous clearing of a traumatic cataract. Jean Sédan and Simone Sédan-Bauby add some details to the previously published description of the micropuncture of the taut capsule of a cataractous lens.

The experimental transplantation of vitreous from one eye of a rabbit to the other was successfully accomplished in a certain percentage of cases under a painstaking technique by John D. Blum and H. M. Katzin who emphasize the surprising lack of inflammatory reaction of the uvea after this operation.

Diathermy coagulation of wounds in the ciliary region is recommended by Thomas, Cordier, and Michel in preference to other therapy of this dangerous type of injury.

A valuable contribution to the diagnosis of human toxoplasmosis and a detailed description of the eye lesions are given by Franceschetti and Bamatter.

Calmettes, Deodati, and Tabarly suggest repeated small blood transfusions in recurrent retinal and vitreous hemorrhages of young adults. E. Redslob suggests tonoscopic examination in the differential diagnosis of real ophthalmic migraine which is characterized by a retinal hypotension. R. Weekers reports on his results in the treatment of hypertensive uveitis with nonperforating cyclodiathermy. Kalt discusses X-ray treatment in retinal blastoma which he recommends be continued through several years.

The possible hypersensitivity of the carotid and cardio-aortic sinus and the danger of a glaucoma attack in the sudden fall of blood pressure are mentioned by Streiff. M. Rauher describes the late and immediate effects of novocaine on normal and pathologic retinal circulation. Jean Gallois emphasizes the importance of capillary dilatation in the evaluation of intraocular diseases and explains the effect on the retinal elements.

A very original paper by R. Weekers deals with the exploration of visual function by testing the critical frequency of fusion—the smallest amount of illumination per second under which a discontinuous stimulus gives a continuous visual sensation. The measurement of the critical frequency of fusion is an extremely sensitive method and sometimes reveals defects which the campimeter does not show.

Other papers of interest include a study on visual disturbances and retinal changes caused by anoxemia in aviators (P. Dumont, J. P. Bailliart, A. Mercier, and J. Duguet) and a study of the cells in the aqueous (Thomas Verrey).

In another group of essays various aspects of intraocular chemotherapy are described. L. Alearts records the circulation of sulfonamides in the aqueous; A. Dubois-Poulsen and H. Miller observe the therapeutic effect of penicillin injections into the anterior chamber; and W. I. Kapuscinsky recommends the puncture of the anterior chamber as a most powerful vasodilatator of the retina in toxic neuritis and optic atrophy.

G. Renard and P. Bregeat discuss their first eight cases of retinitis pigmentosa as treated with subconjunctival implants of placental tissue. They could not notice any change in the appearance of the fundus. Visual acuity was slightly improved in three cases; visual fields slightly enlarged in four; adaptation was subjectively improved in seven.

Many papers carry a detailed biography.

Alice R. Deutsch.

CORRESPONDENCE

Ophthalmic equipment needed*

Editor,

American Journal of Ophthalmology:

This is a great work and the professorship of ophthalmology takes me half time during the year. The rest of the time I am in Mungali. The department is going well but I do need 25 used and fixed-up ophthalmoscopes for my medical students. Do you suppose there are in the profession even more than that good ophthalmoscopes not being used?

So great is the demand and so scarce is the supply that it would be a great lift to ophthalmology in India if we could have here old, used, but good equipment—slit-lamp microscopes, ophthalmometers, instruments of all kinds, charts for examining eyes, just everything that an ophthalmologist needs.

We are doing 1,500 cataracts a year in Mungali and Vellore now and when we do the 10,000 we want to do we will have more work for our fine young, and older surgeons, too.

(Signed) Victor C. Rambo,
Vellore, South India.

Tonometric readings

Editor,

American Journal of Ophthalmology:

If one follows the recent literature relating to the early recognition of glaucoma, one gains the impression that many physicians attach too much importance to small differences in the readings obtained by the use of the tonometer. While efforts have been directed toward enlarging the scale of the tonometer, so that readings can be made with greater accuracy, the inherent sensitivity of the instrument remains the same.

The application of the tonometer in diagnosis is limited by two important factors: (1) The technique of carrying out the measurement is subject to unavoidable errors arising from the personal equations of both patient and doctor, as well as from the nature of the instrument itself; (2) there has been recognized a wide variation in the rigidity and thickness of the coats of the normal eyeball.

These factors, which affect the ability of the tonometer to indent the cornea irrespective of the intraocular pressure, have been analyzed by Friedenwald (American Journal of Ophthalmology, **20**:985, 1937) in an attempt to arrive at a more accurate estimate of the intraocular pressure.

From an historical point of view, it is interesting to note that Schiøtz, himself, was aware that the tonometric reading was influenced by factors other than the intraocular pressure. The following is quoted from his article in the *British Journal of Ophthalmology*, **9**:150 (April) 1925:

"We may safely assume that for each degree of pressure the maximum and minimum deflections in living eyes will be about the same as we have found on dead eyes. . . . With 29 mm. Hg one may have, in different eyes, deflections varying from 2.2 mm. to 4 mm.; with 15 mm. Hg one may have deflections from 6.2 right up to 9.4 mm. Consequently with a deflection of 3 mm. (5.5. gm. weight), the pressure will as a rule no doubt be about 29 mm. Hg, but that deflection might be properly referable to the minimum graph and to an intraocular pressure of 26 mm. Hg, or to the maximum graph and a pressure of 33 mm. Hg. *This should absolutely be borne in mind when using a tonometer: the deflection of the pointer does not indicate a precise pressure but a pressure-range, which for the 5.5 gm. weight is about 6 to 8 mm., Hg, for the 7.5 gm. weight 8 to 10 mm. Hg, for the 10 gm. weight about 13 mm. Hg,* and for the 15 gm. weight about 15 mm. Hg, nor can it be otherwise when we take into consideration

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* Editor's note: This request comes from the Union Christian Medical College at Vellore, South India. The establishment of this college represents one of the most significant developments in the history of missionary enterprise. Actively coöperating in the work of this medical college are 38 mission boards from all over the world and their aim is to establish a medical college to serve all India.

the individual peculiarities in the size of the eye and in the thickness and elasticity of its wall."

From the foregoing, it appears that even if one assumes with Schiøtz that 30 mm. Hg represents the average upper limit of normal tension, one is never certain whether this normal limit may be applied to the eye in question. Thus, a tension of 30 mm. Hg obtained with a 7.5 gm. weight may represent as much as 5 mm. above normal for one eye and as much as 5 mm. below the upper limit of normal in another. Any criteria based on the use of the tonometer must be evaluated in the light of such observations.

(Signed) Adolph Posner,
New York.

PSYCHSOMATIC INTERRELATIONSHIPS

Editor,
American Journal of Ophthalmology:

In his interesting paper on "Psychosomatic interrelationships in ophthalmology" (Am. J. Ophth., **31**:1241 (Oct.) 1948), Dr. Harrington writes: "The prescription of a pair of glasses, no matter what their dioptic power, may result in immediate relief of eyestrain, headache, blurring of vision and the like. Without a true analysis of the underlying cause and its treatment by psychotherapy, however, no more than a transient improvement, at best, can be expected."

One wonders if this is not riding the psychosomatic theory too hard. Every ophthalmologist has daily experience with refraction cases in which the prescription of glasses causes permanent, not transient, elimination of asthenopia or deficient vision or both. That the correction of the refractive error eliminates the trouble caused by the refractive error is a self-evident truth.

Perhaps Dr. Harrington means that it is the emotional conflicts that cause the refractive errors in the first place.

In that case, he should furnish proof that this is true. It is probably true in certain selected cases, but so far as I know, exact data are lacking. The claim is a familiar one. The late Dr. Bates, for example, stated dogmatically that "myopia" results when the patient is telling a falsehood. It is difficult to understand how lying can affect axial myopia even if one disputes established theories of accommodation!

Certainly, the psychosomatic theory offers a fruitful approach. One feels it can be most effective when it is limited to selected cases that are difficult to explain on a strictly organic basis.

(Signed) Philip Pollack, New York.

ABSTRACT DEPARTMENT

EDITED BY DR. F. HERBERT HAESSLER

Abstracts are classified under the divisions listed below. It must be remembered that any given paper may belong to several divisions of ophthalmology, although here it is mentioned only in one. Not all of the headings will necessarily be found in any one issue of the Journal.

CLASSIFICATION

1. Anatomy, embryology, and comparative ophthalmology
2. General pathology, bacteriology, immunology
3. Vegetative physiology, biochemistry, pharmacology, toxicology
4. Physiologic optics, refraction, color vision
5. Diagnosis and therapy
6. Ocular motility
7. Conjunctiva, cornea, sclera
8. Uvea, sympathetic disease, aqueous
9. Glaucoma and ocular tension
10. Crystalline lens
11. Retina and vitreous
12. Optic nerve and chiasm
13. Neuro-ophthalmology
14. Eyeball, orbit, sinuses
15. Eyelids, lacrimal apparatus
16. Tumors
17. Injuries
18. Systemic disease and parasites
19. Congenital deformities, heredity
20. Hygiene, sociology, education, and history

7

CONJUNCTIVA, CORNEA, SCLERA

Bietti, G. B. and Pasca, G. **Research on the activity of streptomycin and its use on the inclusion bodies of trachoma in the conjunctiva.** Boll. d. Soc. ital. di biologia sperimentale. **24**:82-85, Jan., 1948.

Fourteen patients with trachoma were treated with streptomycin and penicillin. Streptomycin has no advantage over penicillin. Improvement was slight and late. I. E. Gaynon.

Bietti, G. B., and Pasca, G. **Use of paraminobenzoic acid in trachoma.** Rev. internat. trachome. **25,** Jan., 1948.

In eight cases of acute trachoma and six of chronic trachoma treated with 25-percent paraminobenzoic acid drops were given every two hours for ten days. Two patients were also given the same drug orally. There was no change in the number of inclusion bodies found. After the use of 0.1-percent sulfathiazole ointment the microscopic manifestations of trachoma disappeared. I. E. Gaynon.

Glees, M. **Clinical picture of smoke damage to the eye.** Klin. Monatsbl. f. Augenh. **113**:43-44, 1948.

This short article discusses the various grades of conjunctival and corneal inflammation after smoke damage. The most malignant corneal lesions were seen in those in whom anaesthetic drops or ointments had been used.

Max Hirschfelder.

Harms, H. **The technique of penetrating corneal transplantation.** Klin. Monatsbl. f. Augenh. **113**:59-64, 1948.

Following the idea of Vannas the author uses condom rubber of the size of a cornea to hold the transplant in place. The rubber is anchored by sutures in the episclera. A manual trephine is preferred to the motor driven type, as it permits more precise control. In order to obtain discs of equal size the pressure should be approximately the same in the donor and in the recipient eye. The danger of injuring the lens and iris is avoided in the following way. The trephine is rotated until aqueous is lost. Instead of completing the incision with scissors rubber is introduced into the anterior chamber as a protection to the underlying structures. The completion of the circular penetration is then continued with the hand trephine which will not penetrate the rubber in the

anterior chamber. The rubber is then removed through the completed trephine hole. The rubber is sterilized by a two-minute suspension in ether and in alcohol solutions ranging from 96 down to 15 percent. (References.) Max Hirschfelder.

Hartman, K. **Ectogenous conjunctival bovine tuberculosis and its treatment by electrocoagulation.** Klin. Monatsbl. f. Augenh., **113**:20-28, 1948.

Infection in a young girl who had handled a dead rabbit is described. The patient exhibited papillary, partially ulcerated granulations of the conjunctiva of the upper lid with severe swelling of the parotid, submaxillary and regional glands. Laboratory tests showed bovine tuberculosis and one excised gland showed caseous necrosis. The persistent conjunctival granulations were removed by electrocoagulation in six sittings. The literature of the Parinaud syndrome is discussed briefly and the author feels that the clinical diagnosis of Parinaud's disease should be superseded by more specific etiologic terms. (References.)

Max Hirschfelder.

Maschler, J. **A case of pyocyaneus ring abscess of the cornea treated with streptomycin.** Brit. J. Ophth. **32**:426-428, July, 1948.

A foreign body imbedded in the cornea of a 15-year-old boy was removed after two days. Three days later there was an extensive abscess in the cornea accompanied by hypopyon. Culture revealed B. pyocyaneus. He was immediately given 100,000 units of penicillin. It was injected under the conjunctiva and dropped into the anterior chamber after paracentesis. Heat, atropine and sulphadiazine were also prescribed. After a few hours streptomycin was instilled into the eye hourly, given intramuscularly every three hours and injected under the conjunctiva once, a total of one gram daily. During one paracentesis it was dropped into the anterior chamber (2,500 units in 2 drops). A broad Saemisch section was done and after three days the whole process regressed completely. After a few days an intense secondary glaucoma occurred and was relieved by a broad basal iridectomy. The central corneal area remained moderately opaque. Morris Kaplan.

Paton, R. T., McLean, J. M., Castroviejo, R., Maumenee, A. E., Kornblueth, W., Owens, W. C., and Woods, A. C. **Symposium: corneal transplantation.** Tr. Am. Acad. Ophth. pp. 312-353, March-April, 1948.

Paton discusses the selection of cases. A patient with reduced vision as a result of scarred cornea, but well adjusted and self supported, might better be left alone even though the corrected visual acuity is below 20/200. Highly nervous patients and those who are poorly adjusted emotionally make especially poor candidates for this type of surgery. Corneal grafting is done in much younger patients than cataract surgery, and it is well to remember that failure in them leads to more serious consequences from an emotional and economic point of view. No eye with scarred cornea should be operated on if iridectomy would bring about an improvement of vision. The most favorable cases are those in which there is a central nebulous opacity in an otherwise normal eye. There must be no active corneal disease, no infection of conjunctiva, cornea, or uvea, no increased tension, and no vascularization of the cornea. Blood staining of the cornea and conical cornea are favorable lesions, but in conical cornea it is not advisable to graft if the vision can be corrected to 20/200 even with a contact glass. Most cases of corneal dystrophy are not favorable for corneal transplantation. Calcareous degeneration, if it involves the entire cornea and full thickness, should not be operated on, but

if it is bandshaped with some clear corneal tissue in the periphery, transplantation should be attempted. Densely scarred corneas from powder burns do not do well. In general tattooing and corneal transplantation do badly in a highly vascularized cornea. There is perhaps no more favorable treatment of descemetocele than corneal transplantation.

McLean describes the process. Local anesthesia supplemented by akinesia and retrobulbar block is preferred. Donor eyes should be enucleated within six hours after death, and preserved at 4 degrees C. in a sealed container having a saturated water vapor atmosphere. Both the square and circular types of transplant operations are described and illustrated. Beveling of the edges of the graft on the recipient cornea is not necessary. Present methods are all variations of the overlying suture to hold the graft in place. The shape of the graft is not of great importance. The high-speed mechanical trephine is the best means of cutting a true-edged graft. When cutting a graft by hand it is easier to make a square one. The operation can be mastered by any competent intraocular surgeon, but it should not be attempted by one who has not had considerable experience in developing his technique through many animal grafts.

Castroviejo describes complications that may occur during the operation and in the period of postoperative recovery. Partial detachment may be resutured, though usually unsuccessfully. If necessary, iris hernias should be excised about the third week and synechiae, the second week. Infection is best combatted with subconjunctival penicillin, streptomycin, and sulfadiazine. In complicating glaucomas, miotics, heat, and, if necessary, sclerectomy are advised. Excessive vascularization is best treated about the fourth week with 1,500 r. of radiation. Excessive and degenerative inflammations may justify the removal of focal infective process with or without intravenous injections of amino acids. Excessive edema may be reduced with 15-percent hypertonic sodium chloride jelly applied 3-6 times daily. Excessive protrusion of the implant is best treated with a properly applied pressure bandage.

In a section on physiopathology Maumenee and Kornblueth describe their experiments which were designed to elucidate the process of regeneration. The wound is closed at first by a fibrin clot, and later by fibroblasts which form fibrous tissue between the graft and the adjacent cornea. The epithelium is replaced by a migration of epithelium from the recipient cornea. The endothelium may be replaced from uninjured cells of the graft, or by migration of cells from the recipient cornea. The majority of the stromal cells continue to live in the graft and do not show massive degeneration or replacement at any time. They are supplemented to a limited extent by newformed keratoblasts from the macrophages of the recipient cornea.

Owen presents a statistical analysis of the results of 417 corneal transplant operations. The graft remained clear in 36.5 percent of the cases. The best results were obtained in keratoconus, hereditary dystrophy, interstitial keratitis and nonspecific inflammatory scars. No grafts remained clear in cases of Fuchs's dystrophy. The visual results after corneal transplantation in patients with preoperative vision of 20/100 do not warrant the risk of operation. Chas. A. Bahn.

Pines, N. **Denig's operation for trachomatous pannus.** Brit. J. Ophth. **32**:385-394, July, 1948.

In this operation, mucosa of the lip is substituted for the scarred conjunctival mucosa. Under local anesthesia the bulbar conjunctiva is cut at the limbus to make a wound one and one half times as

long as the width of the pannus. It is important that this section go down to the episclera. All tissues are removed and the episclera is layed bare. While the assistant is securing hemostasis with hot saline sponges, a special forceps is applied to the lip and a section of the mucosa is cut out slightly longer and wider than the conjunctival wound. This graft is dropped in saline solution and the lip wound closed with one uninterrupted suture. The graft is laid out on a piece of glass and all the fat removed from it after which it is laid into the conjunctival wound with the mucosal side up and stitched with fine silk on three sides. The corneal side remains free. After five days the sutures are removed. At that time the graft will be pink and somewhat edematous if the operation has been successful; it will be white and freely movable if it has not.

The improvement in the eye is usually striking. However, the operation is applicable only in the third and fourth stages of the disease. There is a difference of opinion as to what brings about the improvement. Some observers believe that the peritomy alone is sufficient while others feel that the new tissue is a mechanical obstruction to the progress of the disease. The author presents four case reports. Morris Kaplan.

Rizk, K. **A case of keratomalacia cured by penicillin and vitamin A.** Brit. J. Ophth. **32**:416-419, July, 1948.

A 2-year-old infant developed very severe keratomalacia of both eyes after a brief fever. Both corneas were almost completely opaque, bulged markedly and were covered by deep ulcers. There were few signs of inflammation and the baby seemed to be in no pain. The disease is primarily an avitaminosis A and is not ordinarily considered to be an infectious process. However, penicillin instillations and penicillin in wax were given in large doses. The opacities and the ulcers began to heal almost instantly although the epithelium was slow to regenerate. As soon as vitamin A was given healing progressed rapidly and completely. Dense leucomas remained only in small central areas. Both corneas resumed their normal shapes. Morris Kaplan.

Sjøgren, Henrik. **Keratoconjunctivitis sicca and chronic polyarthritis.** Acta Med. Scandinav. **130**:484-488, 1948.

The author quotes Stenstam's statistics on the occurrence of this ocular condition to show that it exists in 10.5 percent of the cases of primary chronic arthritis. Sjøgren in his studies found that 93 percent of his cases occurred in males; Stenstam found the disease equally divided among the two sexes, yet further along in the article Stenstam is said to have found the severe form of keratoconjunctivitis sicca to be much more prevalent in women. Gifford in 1943 was said to have found one man to eleven women affected by the severe form. The author feels that the diagnosis of a mild case is rather difficult especially since Schirman himself says that his tear-function test is only a "very rough method," one which has never been standardized. As in the Gifford report, Sjøgren, too, found that in 40 percent of his pronounced cases no arthritis was present. The cause of this disease is unknown but two factors are worthy of consideration: one, infection, and the other, endocrine disfunction.

Francis M. Crage.

Stephenson, R. W. **Phlyctenular keratoconjuntivitis treated with penicillin.** Lancet **2**:144-145, July 24, 1948.

The author reports a case of phlyctenular keratitis caused by an allergic reaction to staphylococcus. Response to penicillin therapy was good. I. E. Gaynon.

Swan, K. C., Emmens, T. H., and Christensen, L. **Experiences with tumors**

of the limbus. Tr. Am. Acad. Ophth. pp. 458-469, May-June, 1948.

The diagnostic differences of unusual limbal neoplasms are discussed. Benign leukoplakic lesions are nonvascular and carcinomatous lesions contain new or enlarged blood vessels. The transition to malignancy may therefore be observed by the appearance of new blood vessels. Lymphangiomas of the limbus tend to grow steadily. They do not metastasize but are locally invasive and destructive. They should be completely removed in the earliest possible stage. Simple lymph cysts tend to enlarge especially if they are near the palpebral fissure, and should be excised and not incised. Malignant melanotic tumors commonly arise from precancerous melanomas rather than nevi. Extensive surgical procedures are imperative. Granulomatous lesions may contain foreign bodies. It should be explained to the patient that the excision of lipodermoidal tumors frequently leaves extensive scar tissue. In the removal of limbal lesions a standard laboratory stereoscopic dissecting microscope is preferred. Stretching of Tenon's capsule may reduce or eliminate the need for mucous membrane grafts. Chas. A. Bahn.

Velhagen, K. **The type of vascularization in the differential diagnosis and clinical picture of some corneal diseases.** Klin. Monatsbl. f. Augenh. **113**:28-36, 1948.

The differential diagnosis of corneal inflammations leading to deep vascularization is presented. The vessels take a straight course to the corneal center in luetic parenchymatous keratitis and do not show anastomosis. In the so-called sclerosing keratitis, which is usually caused by tuberculous scleritis, deep vessels enter the cornea for a short course, but instead of proceeding towards the corneal center they either form arcs or run straight through the peripheral parts of the cornea. Interstitial keratitis due to tuberculosis has very irregular and anastomosing vessels without a definite direction. They often surround grayish nodules in the cornea like a net. Rosacea keratitis is characterized by single branches or very small groups of vessels which often appear in the lid aperture. Frequently one finds small subepithelial or ulcerated infiltrates at the end of these vessels and some vessels may overshoot the location of the infiltrate. (4 figures, references.) Max Hirschfelder.

Venco, L. and Morone, G. **Some notes on the pathologic anatomy of grafted human cornea after keratoplasty.** Ann. di ottal. e clin. ocul. **73**:221-246, April, 1947.

Histologic examination was made of six corneal transplants removed because of loss of transparency or for other cause from seventy days to one year after grafting. Dominant in the histologic picture was the edema affecting all layers. The epithelium was found to be markedly edematous from imbibition of fluid. Bowman's membrane was absent in two but was preserved in four cases. Since this membrane lacks regenerative power, its preservation points to the survival of the original membrane in the graft. In four cases the stroma showed cellular infiltration and new formation of capillaries; the lamellae were markedly dissociated and irregularly arranged; in two cases the stroma had undergone fibrillar transformation. Descemet's membrane was broken, detached, and rumpled, but was absent in only one case. In every case the endothelium was missing, probably because of handling in preparation of the specimens and because of involutional processes.

No one scheme was found sufficient to interpret the histologic processes observed. Venco and Morone believe they can distinguish four different possibilities.

1. A total transformation, in which the

graft is replaced by fibrous tissue. This occurs especially when the postoperative course is disturbed by such complications as infection, glaucoma, adhesion of iris to graft, and extensive vascularization and infiltration.

2. A subtotal transformation, in which the graft is only partially replaced by fibrous tissue. This occurs especially when the surrounding corneal tissue of the host is leukomatous. Presumably the leukomatous tissue lacks the vitality needed to cause complete transformation. Some of the original elements of the graft are found to persist, but not without losing their normal characteristics.

3. A partial transformation, in which localized opacities form in certain sectors of the graft and other parts remain transparent. An injury to Descemet's membrane in making the graft may result in poor union and thus occasion a localized organization of the stroma. Irregular edges or improper placement of the graft may cause the failure of normal metabolic relations to be established between host and graft at all points of contact. The part of the graft which will suffer most is that which is in contact with leukomatous or other abnormal tissue in the host.

4. No transformation of the tissue of the graft, which remains transparent throughout. This ideal result may be achieved when the postoperative course is perfectly quiet, without any kind of untoward reaction. In such cases the cicatricial ring between graft and host is frequently so fine as to be almost invisible, and there is reason to believe that a great part of the original tissue survives, especially the fibrillar framework.

(Review of the literature, 9 photomicrographs, bibliography.)

Harry K. Messenger.

Wolffersdorff, v. H. **Comparative studies of the course, treatment and epidemiology of epidemic keratoconjunctivitis in Saxony during 1941 and 1944.** Klin. Monatsbl. f. Augenh. **113**:36-43, 1948.

The author gives a detailed clinical picture of epidemic keratoconjunctivitis as it appeared in Glauchau, Saxony, during two epidemics in the early forties. He presents a statistical analysis of an equal number of patients (322) during both epidemics. In 28 percent of the patients there had been major or minor trauma. The 37 patients operated on for cataract showed symptoms of the disease, but did not suffer serious consequences. In addition to 10-percent albucid solution and vitamin A ointment the author used X-ray irradiation, given in doses of 5 to 10 percent HED at 8 to 10-day intervals up to six doses and injections of 10 to 15 cc. own blood intramuscularly. He believes that injections of blood of convalescents properly typed for blood groups should be further investigated.

Max Hirschfelder.

8

UVEA, SYMPATHETIC DISEASE, AQUEOUS

Bernstein, M. E. and Burks, B. S. **A note on iris rim color and width.** J. Heredity. **39**:93, March, 1948.

Rim color of the iris falls into nine classes: two shades of blue, six shades of gray and one of very dark. In 78 percent of the people studied, the color of the main part of the iris was the same as that of the rim. If one parent had a narrow rim, the incidence of the narrow rim in the offspring was increased.

I. E. Gaynon.

Braun, R. **Tuberculosis and focal infection as causes of iridocyclitis.** Klin. Monatsbl. f. Augenh. **113**:9-20, 1948.

It is pointed out that the importance of tuberculosis as cause of acute and chronic

iritis has been overemphasized in the German literature. The various pertinent clinical and laboratory findings as well as animal experiments are critically discussed. The proof for a hematogenous spread of tuberculosis to the eye is lacking except in the case of miliary tubercles. Much points to the correctness of the American idea of focal infection. Iritis, rheumatism and arthritis may be the result of the sensitization of mesenchym tissues and an allergic response to the primary focus. This focus can be in the tonsils, teeth, gallbladder, pelvic organs and, in some cases, can well be a tuberculous hilus gland. Removal of the focus will not necessarily stop the allergic reaction of the sensitized tissue immediately. Climatic treatment, irradiation, treatment with heavy metals and, perhaps, salicylic acid are helpful in bringing the sensitized tissues back to normal.

Max Hirschfelder.

Gilbert, W. **Early stages of ophthalmia lenta with remarks on the etiology of this disease.** Klin. Monatsbl. f. Augenh. **113**: 1-8, 1948.

Ophthalmia lenta is a most severe chronic eye disease which begins with a recurrent uveitis, but finally affects retina, vitreous and optic nerve. It is always bilateral and is part of a syndrome which includes aphthous stomatitis and ulcerations of the genital organs, particularily of the scrotum. Changes in the skin have the character of an erythema nodosum. Eye symptoms may be so slight in the beginning that the dermatologist is likely to overlook or disregard them. Later, the recurrent severe hypopyon iritis can completely dominate the picture. The author refers to a somewhat similar but much more frequent ophthalmia in horses. He adds two cases to the literature on the subject and expresses the opinion that the syndrome is the allergic response to an infection, possibly by a virus. (References.)

Max Hirschfelder.

Gorduren, S. **Operative treatment of five cases of iridodialysis.** Brit. J. Ophth. **32**:429-435, July, 1948.

Traumatic iridodialysis is not uncommon. It causes cosmetic and functional defects and should be treated as early as possible. The symptoms most complained of are reduced vision, dazzling, and monocular diplopia. Since 1917 the Golowin operation has been most employed and has served quite well. After local anesthesia a keratome incision is made just on the level of the iris root and parallel to its anterior surface. The knife is withdrawn slowly and the iris is allowed to protrude with the aqueous outflow. If the iris does not appear, it is pulled out with an iris hook. With two spatulas the iris is made to rest evenly in the wound and after the instillation of atropine, both eyes are dressed. The patients sit up after the first day, are out of bed the third day and resume work on the seventh day. Morris Kaplan.

Hart, W. W. **Sympathetic ophthalmia. Report of a case.** New Orleans M. and S. J. **101**:85-87, August, 1948.

The clinical history is presented of a 48 year old negro woman in whom sympathetic ophthalmia developed 57 days after a penetrating injury. The inciting eye was enucleated five days later. The sympathizing eye smoldered for another four months and healed with a visual acuity of 20/120. The data presented throw no new light on cause, pathogenesis or treatment. F. H. Haessler.

Hu, Jenner. **Total disappearance of the iris due to essential atrophy.** Am. J. Ophth. **31**:1168-1169, Sept., 1948.

Illingsworth, R. S., and Wright, T.

Tubercles of the choroid. Brit. M. J. pp. 365-368, August 21, 1948.

The authors report their observation on choroidal tubercles in 25 of 42 cases of miliary tuberculosis because of their probable prognostic value, and to emphasize their importance in diagnosis. Choroidal lesions establish a diagnosis before a radiograph of the chest or a lumbar puncture has been performed.

The natural history of the choroidal tubercle is described. As time passes, the margin of the tubercle becomes more definite, the tubercle enlarges, the center becomes paler, and the tubercle and the surrounding area become stippled with pigment. In the final stage there is a parchment-white scar with heavy surrounding pigmentation and paling of the near-by choroid.

It is suggested that further observation may well show that the changes described are of prognostic value. Of those cases (eight in number) in which in spite of prolonged streptomycin treatment the tubercles failed to progress beyond the early intermediate stage, death has occurred in five and the remaining three are doing badly. All the cases in which the tubercle progressed to the parchment-white stage without the appearance of any tubercles in earlier stages were apparently cured. (40 references, 6 figures on colored plate.) F. H. Haessler.

Kapuscinski, W. J. **Tuberculosis and focal infection in diseases of the mesenchymal tissues of the eye.** Arch. d'opht. **8**:238-263, 1948.

Kapuscinski reviews the subject of the pathogenesis of uveitis and discusses in detail the concept, attributed to German authors, according to which the majority of uveal inflammations are of tuberculous origin. He concludes that histopathologic, bacteriologic, clinical, and therapeutic data now available do not substantiate these early claims made for tuberculosis but, on the contrary, tend to give to focal infection the major role in pathogenesis. He supports the view that the uveal tissue is sensitized so that it reacts to antigens released from a distant focus. He mentions the diagnostic value of intravenous injections of typhoid vaccine; a tuberculous infection would tend to be aggravated whereas one due to focal infection would usually improve. Acceleration of the sedimentation rate accompanied by an exacerbation of the uveitis after typhoid vaccine injection would suggest tuberculosis, whereas an acceleration of the sedimentation rate accompanied by improvement in the local focus would suggest focal infection. There is no acceleration of the sedimentation rate when there is no systemic infection.

The author describes 12 cases of which two were undoubtedly tuberculous. The others were a result of focal infection, for the most part dental. He does not support the theory of organotropic bacteria (Rosenow) and concludes that the allergic theory, while more satisfying, does not cover all cases. He concludes by stating that there are only three causes of intrinsic inflammation of the mesenchymal tissues of the eye: (1) syphilis (about 15 percent), (2) tuberculosis (about 3 to 5 percent), and (3) the focal infections (streptococcic, staphylococcic, gonococcic, and others).

Phillips Thygeson.

Lasky, M. A. **Essential progressive atrophy of the iris.** Am. J. Ophth. **31**: 1131-1134, Sept., 1948. (3 figures.)

Marquez, M. **Ocular hypertension in glaucoma.** Ann. d'ocul. **181**:351-361, June, 1948.

Glaucoma and ocular hypertension are not synonymous. That hypertension is only one of the variable symptoms of glaucoma has been shown by the numerous cases of glaucoma without hyperten-

sion which have been reported in ophthalmic literature. The glaucomatous excavation is absolutely independent of ocular tension, and peripheral as well as central visual defects are essentially due to retrobulbar vascular degeneration. Basically, primary glaucoma is always chronic. Primary glaucoma may be controlled but never cured. Among the newer therapeutic agents now under investigation are nicotinic acid and rutin. The use of the former is based on a minimal elective vasodilatation and of the latter, on possible reduction of capillary fragility.

Chas. A. Bahn.

Moutinho, H., and De Moura, S. **Meningococcic iridochoroiditis (an exclusively ocular form of meningococcic infection).** Ann. di ottal. e clin. ocul. **73**: 311-320, May, 1947.

Metastatic iridochoroiditis is a well-known complication of epidemic cerebrospinal meningitis, but the possibility of meningococcic iridochoroiditis without manifest meningitis has hardly been recognized. Moutinho and De Moura present eight cases of unilateral metastatic iridochoroiditis occurring at the height of an epidemic of meningococcic meningitis in children who had no meningitis and were otherwise quite well. The ages of the patients varied from five months to three years. In the evolution of the disease three characteristic stages were recognized: 1. mild general and meningitic symptoms, lasting less than 24 hours; 2. an interval of 2 to 4 days when everything seemed normal; 3. obvious iridochoroiditis. In three patients lumber puncture was done, and in two of these gram-negative diplococci with the morphologic characteristics of the meningococcus were found. The spinal fluid was otherwise normal. An ocular puncture was done in one eye, and the fluid showed diplococci with the same characteristics. The endophthalmitis was severe, with purulent exudate and eventual cicatricial contraction tending to phthisis bulbi.

Harry K. Messenger.

Radnot, Magda. **Contribution to data on sight disturbances caused by proliferation of pigment epithelium.** Brit. J. Ophth. **32**:423-426, July, 1948.

In an 82-year-old woman, who had had an intracapsular cataract extraction with a round pupil several years before, the vision was limited to the counting of fingers and the entire pupil and coloboma was filled with a dark brown film. Slit-lamp examination revealed a solid sheet of pigment on the anterior surface of the vitreous. Presumably the pigment epithelium of the iris had been injured in the operation and had proliferated until it covered the whole anterior surface of the vitreous. Morris Kaplan.

Sautter, H. **Some interesting findings on the iris.** Klin. Monatsbl. f. Augenh. **113**:44-51, 1948.

Two malignant melanomas were cured by iridectomy. A third case is one of a metastatic carcinoma carried from the testis to the anterior uveal tract through the anterior ciliary vessels. Metastasis through the anterior ciliary vessels is relatively rare. A fourth eye is described in which hypoplasia of the dilator of the iris was found in an excised piece of iris. The pupil had not dilated well before cataract surgery. (6 figures, references.)

Max Hirschfelder.

9

GLAUCOMA AND OCULAR TENSION

Bietti, G. B. **Application of carbon dioxide snow in the region of the ciliary body as a means of lowering the intraocular pressure.** Atti del 36 congressa della Soc. oftal. ital. 9, 1947.

In 15 cases of secondary glaucoma carbon dioxide snow was applied to the bulbar conjunctiva in the region of the

ciliary body. Hyperemia is present during the first hour and is followed by a fall in tension that gradually returns to normal in three to six days. I. E. Gaynon.

Bietti, G. B. **Antiglaucomatous operations in trachomatous eyes.** Boll. d'ocul. 27:129-138, March, 1948.

In the Pavia University Eye Clinic, the occurrence of trachoma patients suffering from glaucoma is not rare and even case abstracts illustrate some of the difficulties involved. Five sclerecto-iridectomies, eight trephinations, and one iridectomy were performed. In five eyes the intraocular pressure returned to normal after fistulisation; in six remaining eyes, the pressure was either not lowered or returned to high values after a transient lowering. Anatomic changes may explain this unsatisfactory result: thickening of the conjunctiva, adhesions between conjunctiva and sclera, frequency of hemorrhages, and the postoperative inflammatory reaction in the conjunctival and subconjunctival tissue. These changes make the filtration of the scar more difficult or impossible. Larger conjunctival flaps, larger corneoscleral openings, careful attention to bloodvessels will avoid these failures. Cyclodialysis, diathermocoagulation, and cryotherapy of the ciliary body are the procedures of choice. (1 table, 17 references.)

K. S. Ascher.

Curran, E. J. **Advances in the management of glaucoma.** Postgrad. Med. 3:399-409, June, 1948.

Chronic simple glaucoma is an anatomic defect in the eye which causes it to be functionally unable to meet the requirements of normal production and exit of aqueous. Miotics should be tried. If repeated field studies reveal a diminution, surgery should be resorted to. The various operative procedures are discussed. Iridotomy is believed to be superior to iridectomy. If iridotomy is not sufficient, an Elliot trephining of the sclera with iridotomy is advocated. I. E. Gaynon.

Esterman, Benjamin. **Headache as a symptom of chronic glaucoma.** M. Cl. North America pp. 839-844, May, 1948.

The headache of chronic simple glaucoma is usually supraorbital or frontal, and tends to come on in the evening or in dim illumination. Occasionally it is present on arising and lasts less than one hours. It is most common at the age of marked presbyopia and the symptoms may be induced by such vasomotor stimuli as heat, cold, and strong coffee.

I. E. Gaynon.

Magitot, A. **Provocative tests in glaucoma.** Ann. d'ocul. 181:338-350, June, 1948.

Provocative tests are not of great practical value. At best they give presumptive evidence, especially if positive. Atropin, homatropin or other mydriatics and cycloplegics are not always followed by increased tension in primary glaucoma. Exposure to dark for 30 to 60 minutes frequently increases tension in the glaucomatous for about one hour. Pupillary dilatation and altered pituitary melanophore function are apparently causative. Caffeine administered parenterally or orally was followed by increased ocular tension in 43 percent of a series of normal subjects and by diminished tension in 27 percent. In the glaucomatous the percentages were only slightly different. Although bodily intravenous pressure parallels intraocular pressure, increased intravenous pressure of the head does not aggravate glaucoma. Increased intravenous pressure may be produced by lowering the head, cessation of breathing, stricture of the neck, or by placing an extremity in ice water. The ingestion of large amounts of water preferably on waking, was followed by increased ocular

tension in 82 percent of a series of patients with primary glaucoma.

Chas. A. Bahn.

Schmerl, E., and Steinberg, B. **Central control of intraocular pressure by active principles.** Am. J. Ophth. **31**:1097-1101, Sept., 1948. (3 tables, 1 figure.)

10

CRYSTALLINE LENS

Bietti, G. B., Nižetić, B., Jr. **Tissue therapy of Filatov in degenerative chorioretinitis.** Atti del 36 congressa della Soc. oftal. ital. 9, 1947.

Twenty-five patients with various types of degenerative chorioretinitis were treated with placental extract, codliver oil and aloe. Of these 11 had improved objectively, nine subjectively, two became worse and three were unimproved. The annular scotoma was reduced in two patients and the visual field increased in ten. (1 table.) I. E. Gaynon.

Cordes, F. C. **Surgery of congenital cataracts.** Am. J. Ophth. **31**:1073-1084, Sept., 1948. (6 figures, 35 references.)

Guyton, J. S. **Prevention and management of complications of cataract extraction.** Am. J. Ophth. **31**:1085-1096, Sept., 1948. (1 figure.)

Lancher, A. J. **Cataract surgery in the young diabetic.** Surg. Clin. North America pp. 508-512, April, 1948.

Two patients with bilateral senile cataract, one a women aged 34, the other a man of 46 years were operated on after adequate treatment with insulin. In the first an extracapsular extraction was performed, in the second the intracapsular technique was successfully carried out.

Proper preoperative care and tight section closure by corneoscleral sutures should make the diabetic no greater risk than other patients. Francis M. Crage.

Rossano, R. **Total lens extraction by massage.** Ann. d'ocul. **181**:364-372, June, 1948.

Preliminary corneal massage softens the eye and thereby apparently facilitates lens extraction. It is accomplished by means of a Daviel spoon. Most of the force is applied at the lower limbus. The usual cataract surgery then follows.

Chas. A. Bahn.

Theobald, G. D., and Haas, J. S. **Epithelial invasion of the anterior chamber following cataract extraction.** Tr. Am. Acad. Ophth. pp. 470-481, May-June, 1948.

This malignant complication occurs approximately once in every one thousand cataract extractions. In the series of 14 cases discussed, implantation most frequently occurred because of the inclusion of a tongue of conjunctival or corneal epithelium in a technically imperfect wound. In five cases epithelization was actually traced through the wound, and it could probably have been traced by means of serial sections in the others. Delayed healing occurred with severe chronic iritis in eight eyes. Four were diagnosed clinically by a thin white line on the posterior surface of the cornea. This complication can be prevented by a limbus based flap, preferably concentric with the limbus which would adequately cover the wound with an intact layer of conjunctiva. Chas. A. Bahn.

11

RETINA AND VITREOUS

Belmonte González, Nicolás. **The pathogenesis of hypertensive retinopathy.** Arch. Soc. oftal. hispano-am. **8**:478-487, May, 1948.

Ocular fundus, blood-pressure and retinal vascular pressure, urinalysis, blood urea, visual acuity, and the arterio-venous index were studied in 116 hyperten-

sive patients. Without giving the details of the study, the author concludes that stellar retinitis, with edema, exudate, and hemorrhage, represents the specific response of the retina to anoxia. Anoxia may be caused by a constriction of the afferent vessels, an impediment to the venous return, lack of oxygen in the blood, and intoxication of the retinal cells which abolishes their capacity to use the available oxygen. Before the appearance of a hypertensive retinitis the imminence of anoxia is indicated by a relative retinal hypertension. Ray K. Daily.

Biro, I. **The present state of the problem of retinitis pigmentosa.** Brit. J. Ophth. **32**:411-415, July, 1948.

Although retinitis pigmentosa is most probably caused by disorders of the pituitary gland and to the surrounding diencephalon, it is not a single disease. Two groups are recognized: the inherited and the autonomous; the former may be dominant and recessive. The great majority of the inherited cases are recessive. The autonomous cases are secondary and follow the exanthemata, typhus, malaria, trauma, inflammations, vaccination, persistant vascular spasms and irregularities of nutrition. The author states that there is a clinical difference between the two types but fails to describe that difference.

The outlook for the inherited type is hopeless while that of the secondary type is more encouraging. The author believes that the work of Filatov should receive attention. In 1945 Filatov wrote of treating 110 cases of retinitis pigmentosa with "tissue therapy" but failed to publish the results of these experiments.

Morris Kaplan.

Budde, H. **Cure of a glioma retinae by radium.** Klin. Monatsbl. f. Augenh. **113**: 51-53, 1948.

A one-year-old child had bilateral glioma. The eye with the more advanced tumor was removed and the orbit treated with 280-percent HED X ray filtered through 1 mm. of copper. The other eye was subjected to radium treatment with a total of 300 mgh given in two sittings. The tumor subsequently became smaller and only a big white scar was seen in the fundus six years after treatment. An irradiation cataract developed and had to be removed three years later. Vision is now 5/15. A short survey is given of the gliomas treated by X ray or radium, that have been reported. (References.)

Max Hirschfelder.

Croll, L. J., and Croll, M. **End results in retinal detachment.** Am. J. Ophth. **31**: 1107-1121, Sept., 1948. (1 table, 16 figures.)

Fieandt, von O. **Experiences with transplantation of human vitreous.** Ophthalmologica **15**:257-268, May, 1948.

The author has performed five vitreous transplantations on four human eyes with persistent, heavy posttraumatic vitreous opacities. Normal vitreous was obtained from normal human eyes a few hours after death. A strictly sterile surgical technique was observed during the removal of the vitreous from the donor's eye. The remarkable feature in the author's small series is the occurrence of a severe inflammatory reaction in two of his cases which caused atrophy bulbi in one and led to enucleation in the other case. To explain these severe reactions Fieandt following the suggestion of Hobart (Arch. of Ophth. 10:237, 1933) tested some patients for sensitivity to their own vitreous proteins. The results were equivocal. To the reviewer it seems that the possibility of a bacterial inflammation should have received more serious consideration. Only one of Fieandt's patients derived some definite visual benefit from the transplantation. There is no indication whatsoever that aspiration of

cloudy vitreous and its replacement with sterile saline solution would not have led to the same result. Peter C. Kronfeld.

Gorduren, S. **Six cases of scintillatio albescens.** Brit. J. Ophth. **32**:435-439, July, 1948.

Six cases of snowball opacities of the vitreous are briefly described. The endogenous deposits in the vitreous are very rare and have been reported in 32 of 82,732 patients and in 40 of 65,000 patients. Two types are recognized: asteroid hyalitis which consists of white spherical bodies in normal vitreous, and synchisis scintillans in which sparkling gold particles appear in a fluid vitreous. It is essentially a disease of old age, and almost always unilateral. The bodies are composed of calcium soaps and are said to be associated with general debilitating diseases but this was not the case in this series. Five of these six had incipient or immature cataract in both eyes.

Morris Kaplan.

Jonkers, G. H. **Seasonal variations in the occurrence of retinal detachments.** Ophthalmologica **115**:308-315, May, 1948.

A publication by R. Weekers (Ophthalmologica 110:218, 1945) caused the author to investigate whether or not the onset of retinal detachment was in any way seasonally influenced. The records of retinal detachment in 836 patients with definite histories concerning the time of onset served as the basis of this study. All these patients had contracted the detachment on Dutch soil and were treated for it at Weve's clinic in Utrecht. In this material Jonkers found the same seasonal peak in June that Weekers had observed in Belgium. Peter C. Kronfeld.

Jonkers, G. H. **Seasonal variations in the occurrence of retinal detachments.** Ophthalmologica **115**:316-318, May, 1948.

In the material of the Eye Clinic of the University of Bern no significant seasonal influence could be recognized in the occurrence of spontaneous retinal detachment. This conclusion was based on the history and findings in 197 patients with retinal detachment. Similar studies in Belgium (Ophthalmologica 110:218, 1945) and in Holland (Ophthalmologica 115:308, 1948) did suggest a seasonal factor. Peter C. Kronfeld.

Lloyd, R. I. **Senile macular lesions.** Am. J. Ophth. **31**:1135-1146, Sept., 1948. (15 figures, references.)

Lijó Pavía, J. **Detachment of the vitreous in chorioretinitis, and alterations of the macula.** Rev. oto-neuro-oftal. **23**: 25-29, April-June, 1948.

The author describes and discusses a case of chorioretinitis with cicatrix and macular changes, in which he was able to discern a "hole" in the posterior surface of the vitreous at the site of the fundus lesion, and posterior vitreous separation. Drawings and stereophotographs of the fundus clearly describe the lesions. (4 figures, bibliography.) Edward Saskin.

Riddell, W. J. B. **Detachment of the retina.** Medical Press **219**:323-325, April 14, 1948.

The short review of the subject is one of the articles in the series of "Modern treatment in General Practice." It concerns itself with the etiology, symptomatology, ophthalmic examination, and operative measures employed in the disease. Much credit belongs to Gonin for his painstaking work. There have been too many good results following surgery to assume a pessimistic attitude in the prognosis of retinal detachment.

Francis M. Crage.

Rosengren, Bengt. **The value of anticoagulant drugs in thrombosis of the retina.** Acta Med. Scandinav. pp. 212-217, Suppl. 206, 1948.

In the treatment of six cases of tributary venous thrombosis with dicumerol, thrombolysis occurred in three about four weeks after the therapy was started. The percentage of successes in these cases undoubtedly is reduced where vascular sclerosis and compression from without exist. The age range in the author's cases was 57 to 76 years. Francis M. Crage.

Silván López, Fernando. **Grouped pigmentation of the retina.** Arch. Soc. oftal. hispano-am. **8**:448-456, May, 1948.

Two cases are reported. One patient had a negative family history and no other anomalies except the retinal pigmentation, the other had a number of associated congenital anomalies, traced through three generations. A 15-year-old boy had twelve groups of slate-colored foci in the inferior nasal sector of the left fundus. They were darker near the disc, and less pigmented further away. The number of foci in a group varied from a solitary patch close to the disc to 15 or 20 small ones in the periphery; the majority of the groups contained two or three. Each group gave the impression of having been formed by the fragmentation of a single focus. The contours of the pigment spots were well defined, and some had a fine granulation at the edges. The pigmented areas obscured the choroidal network, and the retinal vessels passed over them without any alteration in their level. Visual acuity and fields were normal. The other patient, 10 years old, with strabismus and amblyopia of the left eye, had three groups of slate colored foci in the fundus of this eye. They were in the horizontal meridian on the nasal side, eight disc diameters from the disc. This patient was of a hyperexcitable temperament, and in addition to the retinal pigmentation, had syndactyly of the toes and a misshaped auricle. His maternal grandmother was blind from glaucoma; the paternal grandfather was a drunkard. The father and two brothers had the same anomaly of the ears, and one brother had syndactyly of the toes. The pathogenesis of this anomaly is discussed. (2 figures). Ray K. Daily.

Soloff, L. A., and Bello, C. T. **The relationship of retinal hemorrhages in hypertensive patients to cerebral hemorrhage: a comparison of the retinal picture in hypertensive individuals who died of heart failure with those who suffered a cerebral hemorrhage.** Am. J. M. Sc. **215**:660-664, June, 1948.

A review was made of the records of all patients with hypertension who had been admitted to Temple University Hospital between 1941 and 1946 and had died or had cerebral hemorrhage. It was found that retinal hemorrhages occurred in 77 percent of 18 patients who died of cardiac failure without cerebral hemorrhage, in 29 percent of 17 patients who had a cerebral hemorrhage, and in patients who had large regions of spasm with or without a marked sclerosis. Retinal hemorrhages cannot be used as a prognostic sign of future cerebral hemorrhage, as they occur more frequently in those who ultimately die of cardiac or renal failure without a massive cerebral hemorrhage. (6 tables, references.)

Bennett W. Muir.

Vetter, J. **Retinal detachment in a case of retinitis pigmentosa.** Klin. Monatsbl. f. Augenh. **113**:53-59, 1948.

The combination of pigment degeneration of the retina with detachment is very rare. The case of a young man is described who developed a detachment in one eye and was successfully treated by repeated electrocoagulation. The pigmentary degeneration advanced after this operation and, after nine years, had led to tubular fields in both eyes. The author points out that in spite of the supposed coalescence of retina and pigment epithelium in pig-

mentary degeneration the union does not seem strong enough to prevent hole formation and detachment in predisposed retinas. This is particularly true in the very early stages, when sprouts from the pigment epithelium have not yet invaded the retina. The relatively good result in the author's patient seems to recommend detachment surgery, even if the detachment is complicated by an existing retinal pigment degeneration. (References.)

Max Hirschfelder.

Weekers, R., and Roussel, R. **The critical flicker frequency after successful operations for retinal detachment.** Ophthalmologica **115**:297-308, May, 1948.

In previous papers Weekers and Roussel applied the method of determining the critical flicker frequency to the study of various retinal and optic nerve diseases. It proved to be a very sensitive index of visual function. Application of this method in cases of cured retinal detachment reveals permanent impairment of visual function even when the retina had been detached for only one month. The operation for detachment of the retina is an urgent one. Every postponement lessens the prospects of healing and also the postoperative restoration of visual function.

Peter C. Kronfeld.

12

OPTIC NERVE AND CHIASM

Bruetsch, W. L. **Etiology of optochiasmatic arachnoiditis.** Arch. Neurol. and Psychiat. **59**:215-228, Feb., 1948.

Syphilis is the most common, and otitis media is another well recognized cause. The role of trauma is difficult to understand. Arachnoidal adhesions alone may be responsible for atrophy of the superficial nerve fibers, but anatomical experience tends to show that they do not produce atrophy of the nerves on a large scale. The tendency to report unexplained cases of optic nerve atrophy under the heading of optochiasmatic arachnoiditis is increasing, and in many of these there probably is a primary morbid process within the chiasm and nerves. The full status of this condition is still not at hand. (4 figures, references.)

Bennett W. Muir.

Stansbury, J. R. **Optic atrophy in diabetes mellitus. A report of three cases in one family.** Am. J. Ophth. **31**:1153-1163, Sept., 1948. (6 figures.)

Tetzu, I., Blatt, N., and Silvian, J. **Interstitial optic neuritis consecutive to a furuncle in the nasal vestibule.** Arch. d'opht. **8**:264-269, 1948.

The authors report the case of a man of 55 years with a furuncle in the left nares of four or five days duration, and with swelling of the lower lid. He had been receiving sulfonamide and penicillin therapy. The vision of the right eye was normal but that of the left was reduced to light perception. Fundus examination showed an atrophy of the optic nerve. After two days of observation and local treatment, the furuncle came to a head and was evacuated. The patient was treated with dionin, strychnine, vitamins B and C, cyclotropine, intravenous glucose, cascosan, transpupillary infrared radiation, and ultraviolet light; there was slight improvement in vision over a period of three weeks. After ruling out syphilis and sinusitis as possible factors, the authors conclude that the optic atrophy was caused by the furuncle but fail to explain the rapid development of the atrophy. They review the literature on the subject of pyogenic infections of the face and their orbital and intracranial extensions.

Phillips Thygeson.

Wood, A. C. **Optic neuropathies: a simplified classification and outline for etiologic diagnosis.** Am. J. Ophth. **31**: 1053-1069, Sept., 1948. (20 figures.)

13

NEURO-OPHTHALMOLOGY

Chamlin, Max. **Visual field studies in neurosurgery.** Surg. Cl. North America pp. 432-437, April, 1948.

Field charts showing enlargement of blind spots and homonymous field defects were proved to have provided considerable help in diagnosis of brain tumors. In some cases this evidence was corroborative, in others a stimulant to further studies, and in still others it was the most important finding in diagnosing the existence and location of a lesion. They are of great value in differentiating inflammatory lesions such as optic neuritis and retrobulbar neuritis from non-inflammatory diseases. Francis M. Crage.

Ethelberg, S., and Jensen, V. A. **"Choked disc" in neurosyphilis.** Acta. Psychiat. and Neurol. **23**:49, 1948.

The author finds lumbar and cisternal puncture contraindicated when papilledema is present, especially since a positive Wassermann reaction in the spinal fluid does not give conclusive evidence of the syphilitic origin of a given intracranial neoplasm.

If ventriculography reveals a normal or slightly increased pressure without signs of tumor, we may ascribe the papilledema to cerebral meningitis and call it meningeal papilledema. If there is no sign of tumor, but the intracranial pressure is increased, then it may be very difficult to distinguish ophthalmoscopically between meningeal papilledema and papilledema produced directly by the intracranial pressure. In meningeal papilledema the veins are slightly engorged and exudates or hemorrhages are few or absent whereas we find a typical choked disc with pronounced engorgement of the veins and typical extravasates in syphilitic basal meningitis or where the pressure is due to a large solitary gummatous tumor. Apart from antisyphilitic therapy and fever treatment decompression may be necessary to prevent the papilledema from developing into secondary atrophy. Since the operation is not entirely an indifferent procedure we may watch and wait till the edema develops into a regular choked disc.

If ventriculography indicates tumor, craniotomy should be done in all cases. Most of the tumors are of nonsyphilitic origin, but even if the lesion is gummatous, which is very rare, craniotomy is indicated because the intracranial gumma is resistent to chemotherapy.

R. Grunfeld.

Godtfredsen, Erik. **Pathogenesis of cranial nerve lesions, notably ophthalmoplegias, complicating herpes zoster ophthalmicus.** Acta Psychiat. and Neurol. **23**:69, 1948.

The author reports eight cases of herpes zoster ophthalmicus associated with cranial nerve lesions. The cutaneous extension, severity, duration, and prognosis do not differ from what is seen in other cases without attending neurologic symptoms. The lesion is a radiculomeningitis that proceeds from the Gasserian ganglion in which the herpes virus is localized. In the ganglion cells we find a very pronounced hemorrhagic inflammatory reaction with necrobiosis, necrosis, and consecutive regional meningitis. The inflammation spreads along the first branch of the trigeminal nerve centripetally and centrifugally. Eventually the inflammation reaches the cavernous sinus where it involves the third, fourth and sixth nerves. The involvement of the seventh nerve must be considered a manifestation of brainstem encephalitis. The zoster virus spreads centripetally into the brain stem along the descending root of the trigeminal nerve. The facial nerve fibers and the descending root of the trigeminal nerve are intimately related in the pons. R. Grunfeld.

Klein, R., and Attlee, J. H. **The syndrome of alexia and amnesic aphasia. Subarachnoid haemorrhage with symptom of partial occlusion of the spinal subarachnoid space.** J. Ment. Sc. **94**:59-69, Jan., 1948.

The article describes a complete study of a case of subarachnoid hemorrhage in a women, aged 47 years. Neurologically the disease was followed through coma, mania, confusion, and finally convalescence. The neurologic signs were varied and memory suffered the most. Retinal and vitreous hemorrhages and an upper right-sided homonymous quadrantic hemianopia were the outstanding ocular defects. The latter remained unchanged but practically all the other symptoms disappeared at the end of three months. The field defect was ascribed to a break in the lower radiation fibres. Right-sided hemianopic defects when associated with alexia arise from a left lingual gyrus lesion in a right handed person.

Francis M. Crage.

May, P. R. A. **Pupillary abnormalities in schizophrenia and during muscular effort.** J. Ment. Sc. **94**:89-98, Jan., 1948.

Dilatation of the pupil with impaired light sensitivity has been known to occur after muscular effort and in catatonic schizophrenia where the sign was termed "catatonic pupil." In such cases dilated pupils and impaired light reflex were usually bilateral. The author's investigations were made on 343 male schizophrenics and 100 mentally normal males all under the age of 55. Studies were made of the pupils in respect to their equality and the reactions to pain, light and muscular effort. Diminished pupillary response occurred very frequently with the above mentioned reactions in all of the schizophrenics. These abnormal changes might be correlated with the schizophrenic's lack of normal emotional response to the outside world. The pupillary signs found in these cases would complicate the differential diagnosis between schizophrenia and neurosyphilis.

Francis M. Crage.

Simpson, J. F. **Horner's syndrome due to an osteochondroma of the first rib.** Canad. M.A.J. **59**:152-155, August, 1948.

A case of Horner's syndrome in a woman caused by an osteochondroma of the left first rib is reported. The author believes it to be the first reported case of that nature. Symptoms were apparent at the age of 7 years, the syndrome was first noticed at the age of 14, the tumor was identified at the age of 17, and removed at 34 years of age. A review of anatomy, physiology, and etiology is given. (8 figures, references.) Bennett W. Muir.

Stengel, E. **The syndrome of visual alexia with color agnosia.** J. Ment. Sc. **94**:46-58, Jan., 1948.

Word blindness as a loss of visual memory has been ascribed to a localized cerebral lesion and also to a general impairment of brain function. The two cases presented occurred in men, aged 63, and 68 years of age. Each had a right-sided hemianopia, word blindness, and abnormal color vision. The lesions in both cases were obviously vascular. A discussion of the location of lesions causing this complex follows. Francis M. Crage.

14

EYEBALL, ORBIT, SINUSES

Barnett, H. N., and Ford, R. **Ophthalmic and other affections predisposed to by deformities and caused by sepsis of the nose.** Medical Press **219**:239-245, March 17, 1948.

Deformities in the nasal septum and turbinates are considered the main cause for septic infections in the sinuses. The sinusitis results from the mechanical interference with the discharges from the sinuses. The treatment when surgical

must correct both the sinus lesion and the obstructive defect.

Such ocular conditions as dilated pupil, ptosis, corneal ulcers, keratitis, iritis, choroiditis, retrobulbar neuritis, and optic neuritis have been caused by sinus infections. Illustrative cases are presented. The rhinologist is advised to puncture and irrigate sinuses in disturbances of undetermined etiology, despite negative nasal and X-ray findings. This is especially true in retrobulbar neuritis. Contrary to the conclusion of others, the authors feel that the latter disease can be frequently caused by sinus disease, and, because no definite inflammation is present in the nerve, they feel that the symptoms are the result of pressure on the optic nerve by the orbital edema adjacent to the affected sinuses. They feel that a better term for the disease is "pressure paresis of the optic nerve." A strong appeal is made for a thorough nasal study in eye diseases of unknown etiology. Francis M. Crage.

Fischer, J. T. **Basedow's hyperophthalmia.** Rev. oto-neuro-oftal. **23**:29-35, April-June, 1948.

This paper is a continuation of investigations described in the previous issue. To diagnose this condition, several criteria must be kept in mind. The patient complains fundamentally of his eye and vision, the thyrotoxicosis responds to medication, there is a hypometabolism with or without hypothyroidism, and an increase in thyrotropic hormone. Thyroidectomy is contraindicated. The exophthalmos is a true edematous phenomenon, and is attended by the vices of any proptosis. The condition is abetted by the administration of estrogenic hormones and iodothyroxin; relief may also be obtained by radiotherapy directed to the hypophysis and the orbit. In cases of severe proptosis, decompression of the orbit may be attempted.

Edward Saskin.

Loddoni, G., and Duc, C. **Ageing of the human eye.** Boll. d'ocul. **27**:139-192, March, 1948.

This comprehensive review, consisting of 50 pages of condensed factual reports and two pages of references, can not be abstracted briefly. The anatomic and functional changes of the eye during senescence are described. Scleral hyalinization (Roper) is not mentioned. Intravascular pressure, pupillary changes, visual field defects and ageing of the color sense and of light perception are discussed. K. W. Ascher.

Lloyd, R. S., and Jones, R. M., Jr. **The Cutler-Guyton eye implant and prothesis: two cases.** Military Surg. **101**:205-207, March, 1948.

Two cases in which the Guyton modification of the Cutler implant has been used are described. The implants are well tolerated, there has been minimal exudate and there has been 90 percent motility. The operative technique is described.

I. E. Gaynon.

Morone, G. **Cutaneous fibromatosis with orbital angiofibroma.** Boll. d'ocul. **27**:193-199, March, 1948.

Numerous fibropapillomas of varying size were located on the back and on the chest of a 45-year-old patient who had noticed a slight protrusion of his left eye three years before the operation. Transpalpebral orbitotomy revealed the presence of a fibroangioma. The author did not find an identical case report in the literature but the coincidence of Recklingshausen's disease and various tumors has been reported previously. (4 photographs, bibliography.) K. W. Ascher.

15

EYELIDS, LACRIMAL APPARATUS

Callahan, A. **Reconstruction of the canthi.** Tr. Am. Acad. Ophth. pp. 486-496, May-June, 1948.

The outer canthus is normally higher when the lids are closed, but lower when they are opened. The reëstablishment of posterior pull is of special cosmetic importance. This is normally accomplished medially by Horner's muscle and laterally by the canthal ligament.

Two holes are made in the anterior or posterior crest with a dental drill, avoiding perforation of the nasal mucosa. A stainless steel wire is directed through the upper opening, passes between the bone and nasal mucosa and emerges through the lower opening. The ends are then passed through the canthal ligament or tarsal plates and are twisted rather than knotted. This basic technique is necessarily modified to meet individual problems. A series of 37 patients operated on by this method is analyzed. Numerous specific case reports and 27 illustrative figures are presented.

Chas. A. Bahn.

Callahan, Alston. **Severe lid deformities.** Plast. and Reconst. Surg. **3**:456-463, July, 1948.

The management of bilateral multiple lid deformities is illustrated with a detailed case report. Loss of skin is replaced with a free skin graft, and displaced tissues are rearranged with a pedicle flap. Intermarginal lid adhesions are utilized to prevent contraction. When extensive repair of the lid is required after the eye has been removed, the graft or flap is kept under more adequate tension if an acrylic conformer is kept constantly in the socket. This ophthalmopedic aid also maintains ample space for an artificial eye when surgery is concluded. Intermarginal lid adhesions are divided after three months. A severed palpebral ligament of the upper lid of the other eye is reattached to the bone with wire. (7 photographs, 4 drawings.) Alston Callahan.

García Miranda, Ramón. **Sjögren's syndrome associated with acne rosacea.** Arch. Soc. oftal. hispano-am. **8**:590-602, June, 1948.

A case of Sjögren's syndrome and acne rosacea is reported and the newer literature is reviewed with special reference to hormone therapy. A woman, 58 years old, developed in the course of nine years, Reynaud's disease, keratoconjunctivitis sicca, acne rosacea of the cornea, and a general dryness of the mucous membranes, anosmia, loss of taste, and menstrual irregularity. Her ocular as well as general symptoms improved rapidly on treatment with polyvitamins, liver extract, and perandren. Because of the high cost of perandren, its use was discontinued, and the symptoms recurred in their original intensity. She was then given cortical hormone instead of perandren and she again improved remarkably. The local treatment consisted of sodium bicarbonate eye baths. (2 figures.)

Ray K. Daily.

Kettesy, A. **On genesis and operation of the cicatricial (trachomatous) entropion of the upper lid.** Brit. J. Ophth., **32**: 419-423, July, 1948.

Most operations for entropion of trachoma fail because it is erroneously assumed that the cause of the entropion is the progressively incurved tarsus. Actually this incurving of the tarsus occurs quite late in the process and the real cause is the obliteration of the intermarginal space by the contraction of the scars along the subtarsal sulcus. The author has devised his own operation which aims at the remodelling of the intermarginal surface. The upper lid is everted by Liebermann's sutures and the tarsus is incised through and along the line of scars in the sulcus. Three mattress sutures are placed in the conjunctival side of the section and an incision is made just behind the marginal tarsal strip through to the skin. The mattress sutures are then brought through the

tarsal strip and loosely knotted; then the loose ends are brought out through the skin above and tied. Vaseline pressure dressing is applied and the knots are removed after two days. The author dislikes the use of beads, rolls or pads beneath the skin sutures. (5 illustrations.) Morris Kaplan.

Risdon, Fulton. **The eyelid and socket surgery.** Plast. and Reconst. Surg. **3**:454-455, July, 1948.

Skin grafts of .018 are used to reconstruct either upper or lower eyelids. If all four lids require skin grafting, the upper lids are done at one operation, the lower lids at a second. Intermarginal adhesions have not been satisfactory in the author's hands.

A thin lead plate with an intermediate piece of lint is laid over the graft and sutures tied around it to maintain pressure.

In the reconstruction of a contracted socket, the mucous membrane remaining is not removed and a skin graft of .018 thickness is used to cover the raw surfaces produced by the extension. Dental compound mould is preferred. Glass artificial eyes are preferred. Fascia lata strips are used to suspend ptosed lids to the occipitofrontalis muscle.

Alston Callahan.

Rosas Costa, M. T. **Treatment of styes by intrapalpebral penicillin injection.** An. argent. de oftal. **8**:13-16, Jan.-Feb.-March, 1947.

In order to achieve a quick and efficient therapeutic result in the treatment of hordeola and to prevent any complications, the author advises the usual hot wet compress followed by an injection of 50,000 units of penicillin in about one and a half cc. of 2-percent novocaine. The healing is rapid, there is very little local reaction, no other medication is necessary, and one injection usually suffices. (Bibliography). Edward Saskin.

Zarrabi, M. **The operation of entropion,** Arch. d'opht. **8**:269-270, 1948.

Zarrabi discusses the causes of failures in the surgical treatment of trichiasis and entropion due to trachoma. He describes an operation for which he claims a high percentage of successful results. In his operation two parallel incisions are made in the skin of the lid, the first 2 mm. above the ciliary border and the second 3 mm. above the first. This portion of the skin and of the subjacent tissues down to the tarsus is then excised with scissors. Two parallel incisions are then made in the tarsus 1½ mm. apart, the first incision being made 1 mm. from the lower border, sparing the roots of the cilia. This portion of the tarsus is then excised, the palpebral conjunctiva being left intact. A zigzag suture is then passed through the tarsus and the subcutaneous tissue in such a fashion that when tightened it draws the edges of the tarsal incision together and everts the lid border. The skin itself is not included in the suture. The author states that the operation has been employed on a large scale in Teheran with full satisfaction to patient and surgeon. Phillips Thygeson.

17

INJURIES

Cross, A. G. **The effects of mustard gas on the eyes.** Medical Press **219**:281-282, March 31, 1948.

A short historical summary on the use of this war gas is given.

Acute mustard gas keratoconjunctivitis is characterized by the appearance of photophobia and blepharospasm three to eight hours after exposure to the gas. Moderate to severe pain is present in direct proportion to the extent of destruction of corneal epithelium. About 75 percent of the cases were mild and cleared up in about four weeks. Of the remainder,

15 percent with some roughness of the cornea took a little longer and the other 10 percent required many months to heal extensive necrosis of superficial corneal epithelium.

Delayed mustard gas keratitis appeared in a few cases about ten years after exposure to the gas and freedom from ocular symptoms. All these patients gave a history of original treatment of the eyes for as long as six months. The main symptoms were lacrimation, photophobia, and failing vision. The corneal stroma showed dilated vessels and aggregations of caseous material. Ulceration was also present. The vision progressively diminished despite the treatment. The treatment is that of keratitis in general. Contact lenses, where tolerated, were of considerable help to those with irregular corneal astigmatism. The essential cause for the delayed and recurrent corneal ulceration was the presence of masses of cholesterin and fat resulting from the original vascularization in the cornea and limbal region. Once the degenerative process has begun it continues.

Francis M. Crage.

18

SYSTEMIC DISEASE AND PARASITES

Bergstrand, Hilding. **Hypertensive vascular changes in the eye.** Acta Path. et Microbiol. Scandinav. **25**:98-106, 1948.

Volhard's theory of spasticity of the retinal vessels rather than a reaction from toxic substance as a cause for the condition known as albuminuric retinitis is rather strongly upheld. The author substantiates this by the ease with which this theory can explain such conditions as eclampsia, chronic glomerulonephritis, and essential hypertension with or without renal disorders.

The histological changes in the vessels of the choroid and retina in essential hypertension and retinal changes are described.

Francis M. Crage.

Chandramowli, A. **Ophthalmology in relation to general medicine.** The Antiseptic **45**:433-448, July, 1948.

In this lecture the author deals briefly with a large number of topics. He points out the similarity in the behavior of the eye and other organs to stimulation by drugs, he discusses the medical aspect of eyestrain, and describes ophthalmic manifestations of disease of the blood and cardiovascular system, the organs of respiration, and of diabetes, allergic states, and endocrine disturbances.

F. H. Haessler.

Del Río Cabañas, J. L. **Ophthalmoplegic migraine, caused by a metastatic tumor.** Arch. Soc. oftal. hispano-am. **8**: 622-630, June, 1948.

The literature is reviewed, and a case reported. A 66-year-old man with an intestinal neoplasm and bronchial metastasis developed a typical ophthalmoplegic migraine on the right side after a blow in the right temporal region. The third, fourth, fifth and sixth nerves were involved. The ophthalmoplegia is attributed to metastasis at the base of the brain. (6 figures.)

Ray K. Daily.

Ferrié, J. **Complications of relapsing fever.** Ophthalmologica **115**:227-240, April, 1948.

A severe epidemic of relapsing fever broke out in Morocco in January, 1945 and spread from there all over North and Northwest Africa. Visual complications were observed chiefly after the use of pentavalent arsenicals. A neurotropism of the spirochete may also have been a factor in causing optic nerve lesions. The lesions were chiefly retrobulbar and often associated with signs of polyneuritis. The author warns against the use of pentavalent arsenicals in the treatment of relapsing fever.

Peter C. Kronfeld.

Holland, H. **Hydatid of the orbit.** Brit. J. Ophth. **32**:395-396, July, 1948.

Very briefly the author discusses eight cases of echinococcus cyst of the orbit which occurred in India. In all of them the cyst was in the upper fornix. Proptosis was the most prominent symptom and pain one of the least. Vision was little disturbed unless ulceration of the cornea had developed. It was always unilateral and the diagnosis was made by finding typical hooklets in the fluid obtained by aspiration of the tumor. It is important to completely excise the cyst which is most difficult to do. The author succeeded in two of his patients. Ecchymosis reaction is very severe in patients in whom portions of the cyst remain; however, the prognosis for vision is very good.

Morris Kaplan.

Palmer, R. S., Nyssens, A. F., and White, J. C. **Severe hypertension with papilledema simulating brain tumor.** New England J. Med. **239**:322-327, August, 1948.

The similarity in symptoms, physical signs, and spinal fluid findings in patients with severe hypertension with papilledema and in those with expanding intracranial lesions is pointed out. A comparison is made of observations in 30 patients with severe hypertension and papilledema, in 25 patients with expanding intracranial lesions, and in 15 patients with severe hypertension without papilledema. The differential diagnosis between severe hypertension with papilledema and an expanding intracranial lesion with coincident hypertension is discussed and illustrated by a report of three cases. The most important differential characteristic is the slow, steady progression of neurologic localizing signs in brain tumor. Proper evaluation depends upon expert neurologic examination and sometimes upon supplementary pneumograms. In the hands of the writers extensive sympathectomy has been the measure that relieved symptoms, reduced the papilledema and stayed the usual rapid progress of malignant hypertension. It is suggested that denervation of the splanchnic bed abolishes or minimizes the orthostatic pressor responses and thus protects the cerebral circulation. (3 tables, references.)

Bennett W. Muir.

Rosen, E. **Some new concepts concerning ocular complications following vaccination.** Ophthalmologica **115**:321-332, June, 1948.

Following the mass vaccination against smallpox in New York City during the Spring of 1947 the essayist observed several cases of ocular complication which he reported in various periodicals. They included such syndromes as interstitial keratitis associated with vestibuloauditory symptoms, metaherpetic keratitis, dendritic keratitis, exudative chorioretinitis, central serous retinopathy, iritis and panophthalmitis, thrombophlebitis and lesions of extraocular muscles. In all these cases the eye complication occurred from 9 to 13 days after the vaccination, which period has become recognized as the incubation period of postvaccinial encephalitis. None of the essayist's cases showed definite signs of encephalitis, but in his opinion "it is not difficult to assume that the eye may be the mirror in which allergic cerebral changes encountered in the postexanthematous encephalitic state may actually be visualized." Since postvaccinial encephalitis is a comparatively new disease first described 23 years ago, it is not difficult to understand the infrequency of observation and recognition of the postvaccinial ocular complications. The clinical manifestations of postvaccinial encephalitis are just as variable as the postvaccinial eye complications. The ophthalmologist is in

an advantageous position to observe postvaccinial complications.

Peter C. Kronfeld.

Saebo, J. **Xeroderma pigmentosum with affection ot the eye.** Brit. J. Ophth. 32:398-411, July, 1948.

Xeroderma pigmentosum is a congenital skin disease which is relatively rare and very difficult of diagnosis. It develops under the influence of sunlight in the course of the first few years of life. Three stages are recognizable: the first is a hyperemic and inflammatory phase most often called ordinary freckles; a second stage is characterized by brown warts and excrescences; and in a third stage malignant growths appear. The disease takes many years to develop but most patients die at an early age. Serious ocular manifestations occur often, such as malignant palpebral affections, pigmented conjunctival tumors, teleangiectases and papillomas. They may spread to all parts of the eye and to other parts of the body.

The author describes three cases which were closely watched for many years. The first two were in siblings in whom the disease was discovered early. Each outcropping was promptly treated with heavy doses of X-ray or radium radiation, with good and apparently lasting results. The third case was not diagnosed for many years. Both eyes had to be removed and the patient died with extensive metastases at the age of 21 years. It is important that even benign-looking tumors in the eyes associated with pigmented spots on the skin be studied most carefully. (10 figures.) Morris Kaplan.

Teichman, B., and Greer, K. C. **Ocular manifestations of myasthenia gravis.** Canad. M. A. J. 59:55-58, July, 1948.

The onset is usually gradual and increased muscular fatiguability is most often observed in the ocular muscles. Ptosis of one or both upper lids is the first symptom in over 50 percent of cases, and soon is associated with variable diplopia. Less frequently the bulbar muscles are involved. A characteristic is the increase in signs and symptoms with bodily fatigue, and regression after rest. Orbicularis weakness is sometimes seen. Pupillary reflexes are not interfered with, and accommodation paresis is rare. Visual acuity and visual fields are not involved. Sometimes symptoms remain evident only in the ocular structures for years although usually they spread to other skeletal muscles. Subcutaneous injection of 1.5 mgm. of progstigmine gave improvement of the levator muscle action in all of one series of cases. The muscles attached to the globe, however, may require a larger dose to improve ocular motility. Two doses of 0.6 gm. each of quinine may be used if the progstigmine gives no results. Myasthenia should be included in the differential diagnosis of external ophthalmoplegia. The exact relationship of the thymus to this disease is not known. However, a patient may benefit from thymectomy. (1 figure, references.) Bennett W. Muir.

19

CONGENITAL DEFORMITIES, HEREDITY

Busacca, A., and Pinticart, de W. E. **Gonioscopic study of a case of posterior corneal embryotoxon.** Ophthalmologica 115:283-290, May, 1948.

In 1920 Axenfeld gave the name posterior corneal embryotoxon to a congenital anomaly of the cornea characterized by a white annular band situated in the deepest layers of the cornea just inside the limbus. In the paper under review the clinical and gonioscopic findings in such a case are described in detail and well illustrated. The white band proved to be

an anterior extension of the corneoscleral trabeculum. The glistening white line that formed the inner border of the band was the thickened tip of the scleral septum which is the posterior extension of sclera into the cornea and serves as foundation for the corneoscleral trabeculum and the canal of Schlemm.

Peter C. Konfeld.

Crespi Jaume, G. **Congenital ocular melanosis.** Arch. Soc. oftal. hispano-am. **8**:437-447, May, 1948.

This is a comprehensive review of the literature, and a report of three cases. A child, four years old, had a darkly pigmented zone 4 to 5 mm. wide surrounding the left cornea, except for a small area on the nasal side. Its outer edge was festooned, and the inner edge was on the limbus. The iris was very dark, and the fundus so deeply pigmented as to obscure the perivascular choroidal spaces. A woman, 58 years old, had twelve dark patches in the superior and temporal portion of the right eyeball. They were separated from one another by areas of normal sclera. The iris was densely pigmented, except for one sector below, where its color and structure were similar to that of the left eye. The third patient, 15 years old, had a choroidal nevus, and small pigment spots on the disc. (3 figures.) Ray K. Daily.

Papolczy, F. **Congenital cyclopia and orbital cyst together with other developmental anomalies on the same side of the face.** Brit. J. Ophth. **32**:439-443, July, 1948.

The author describes a baby with characteristics of congenital cyclops confined to the right side. A proboscis-like tube, 3.5 cm. long and 1.5 cm. in diameter, extended from the right inner canthus down along the nose and had a navel-like depression on its distal end. The lower lid was violet and was greatly enlarged into a very soft cyst. There was a hare-lip and cleft palate on the right side and no eyeball. The left side was normal. The proboscis and the cyst were removed. It was found that the tube had all the tissues of the normal nose and the cyst had only layers of epithelium. Eight days after this operation the baby developed pneumonia and died. At necropsy it was seen that the soft parts of the orbit contained tissues of lens, retina, choroid, ciliary body, iris and pigment epithelium.

Morris Kaplan.

20

HYGIENE, SOCIOLOGY, EDUCATION, AND HISTORY

Birren, F. **The ophthalmic aspects of illumination, brightness and color.** Tr. Am. Acad. Ophth. pp. 566-584, May-June, 1948.

In industry, glare and other symptoms result from maladjustment between increased illumination and environmental brightness. Illumination which may appear excessive is frequently made to seem normal by adjustment of surrounding brightness. Flooring materials, desks, filing cabinets, woodwork and paints enter into the adjustment. Fairly critical seeing usually requires 25 to 35 foot candles and general illumination should ordinarily not exceed 50 foot candles. The theory of uniform brightness is both impractical and misleading. The illumination of the work should moderately exceed its surroundings. Monotony, however, should be avoided. The psychologic aspects of vision including color constancy are of special importance. Ocular fatigue is largely muscular rather than retinal. The constant effort to improve vision by ineffectual adjustments causes the symptom complex known as eye strain. Chas. A. Bahn.

Currie, J. G. D. **An English experience.**

Tr. Am. Acad. Ophth. pp. 584-588, May-June, 1948.

In an English factory which produced precision work an excessive number of employees required ophthalmic treatment. A large number of the employees were not visually suited for accurate near work, ametropias were not adequately corrected, and lighting facilities were inadequate. A marked decline in the number of employees requiring ophthalmic treatment was noted when these conditions were corrected. Chas A. Bahn.

Erpf, S. F., Dietz, V. H., and Wirtz, M. S. **Ophthalmoprothesis, U. S. Army World War II.** Military Surg. **101**:405-408, Nov., 1947.

A short history of the development of the artificial eye program is presented. I. E. Gaynon.

Franklin, C. F., Sr. **A bacteriological comparison of penicillin and silver nitrate for prophylaxis against ophthalmia neonatorum.** Military Surg. **102**:179-184, March, 1948.

Two and one-tenth percent of the infants exhibited pus while in the nursery after penicillin prophylaxis and the cultures were positive in 80 percent of these. Six percent of the infants exhibited pus after silver nitrate prophylaxis. Fifty-two percent of the cultures were positive in both groups of infants who exhibited ocular abnormalities other than pus. After penicillin prophylaxis 3.1 percent of the eyes exhibited pus at home and 3.3 percent of the cultures of these eyes were positive. After silver nitrate prophylaxis, 2.4 percent exhibited pus in the eyes at home and 100 percent of the cultures were positive. The gonococcus was found in one case after silver nitrate prophylaxis. I. E. Gaynon.

Koch, F. L. P. **Some practical aspects of geriatric ophthalmology.** Geriatrics **3**: 163-165, May-June, 1948.

Early or impending pathologic changes in the elderly may be determined by examinations. Corrective measures may be instituted at the optimum time. I. E. Gaynon.

Kefalas, A. **Eye hazards and safety practices in the Wearside shipyards.** Brit. J. Indust. Hyg. **5**:51-69, April, 1948.

Of a group of 100,000 miscellaneous accidents 14,722 occurred in the eye. Eye injuries consist of flash injuries, burns or scalds, mechanical trauma, foreign bodies and hot foreign bodies. (10 tables, 7 figures.) I. E. Gaynon.

Morgan, W. G., and Stump, N. F. **Important uses of industrial vision tests and the medical director.** Indust. Med. **17**:253-258, July, 1948.

Employees whose visual skills passed the minimum requirements for the job had greater comfort, simplification of work tasks, increased earning, decreased rejected materials at inspection, greater safety at the job, reduced minimum pay make-up and decreased absenteeism. I. E. Gaynon.

Saunders, E. E. **Navy eye-correction, eye-protection program.** Tr. Am. Acad. Ophth. pp. 562-565, May-June, 1948.

The numerous physical and mental factors that affect the visual efficiency of the individual are discussed briefly. The program instituted by the Navy includes the examination of civilian employees with the Ortho Rater or/and Sight Screener to test simultaneous binocular vision, visual acuity, stereopsis, muscle balance and color discrimination. To facilitate minimal transportation expense, workers are examined preferably in larger key stations and smaller supplementary stations, if necessary. The neces-

sity of occupational glasses in specific cases is stressed and illustrated.

Chas. A. Bahn.

Stewart, R. E. **Plastic artificial eye and restoration program, Veteran's Administration.** Military Surg. 101:396-404, Nov., 1947.

A step by step account of the procedure used in the manufacture of ocular plastic prosthesis is given.

I. E. Gaynon.

Vail, D. **Opportunities and responsibilities of ophthalmology.** Tr. Am. Acad. Ophth. pp. 359-366, March-April, 1948.

In industrial practice ophthalmologists have not fulfilled their duties. In the stress of private practice, most ophthalmologists have not taken the time and effort which is necessary for the reasonable understanding of industrial needs. Problems such as job analysis, illumination in industry, and more efficient industrial methods in the prevention and treatment of employees are important in industrial ophthalmic practice, but unsolved. Generally speaking, ophthalmologists feel that mass refraction means inefficient refraction. The relationship between ophthalmologists and technicians such as optometrists is discussed at some length. Chas. A. Bahn.

Young, M. N. **Ophthalmic practice with an Army Hospital in North Africa and Southern France Ports.** Military Surg. 101:211-212, Sept., 1947.

Ophthalmic practice consisted for the most part of clinic practice comprising refractions, spectacle fittings, physical examinations and consultations. Diseases encountered were keratoconjunctivitis, corneal ulcer and malarial herpetic keratitis. Surgery was performed for orbital fractures, foreign bodies, hordeolum, chalazion, lacerations, pterigium and intraocular foreign bodies. I. E. Gaynon.

NEWS ITEMS

Edited by DONALD J. LYLE, M.D.
601 Union Trust Building, Cincinnati 2

News items should reach the editor by the 12th of the month

DEATHS

Dr. Robert James Lynch, Des Moines, Iowa, died May 28, 1948, aged 67 years.

Dr. Percy Jay Ross, Chicago, Illinois, died September 28, 1948, aged 46 years.

Dr. Vivean Visscher Wood, St. Louis, Missouri, died June 20, 1948, aged 61 years.

MISCELLANEOUS

TORONTO REFRESHER COURSE

Dr. Ramon Castroviejo of New York City and Dr. Lawrence R. Boies of Minneapolis, Minnesota, will be the guest speakers at the combined refresher course in ophthalmology and otolaryngology, University of Toronto, Faculty of Medicine, to be held January 24 to 29, 1949. Dr. Castroviejo will lecture on cataract surgery and give a surgical clinic; Dr. Boies will speak on general problems of interest in the practice of otolaryngology.

GILL GRADUATE COURSE

Gill Memorial Eye, Ear and Throat Hospital presents its 22nd annual spring graduate course in ophthalmology, otology, rhinology, laryngology, faciomaxillary surgery, bronchoscopy, and esophagoscopy, from April 4 to 9, 1949.

Guest speakers include the following: Dr. Banks Anderson, Dr. H. W. Brown, Dr. W. B. Clark, Dr. W. B. Dearring, Dr. W. W. Eagle, Dr. Harold Falls, Dr. E. P. Fowler, Jr., Dr. Leslie Gay, Prof. L. B. Garrod, Dr. Glen G. Gibson, Dr. C. L. Jackson, Dr. Carl C. Johnson, Dr. A. C. Jones, Dr. Harold H. Joy, Dr. Peter Kronfeld, Dr. John S. Lundy, Dr. Louis Panfique, Dr. Peter N. Pastore, Dr. Bernard Samuels, Dr. E. W. Sheldrup, Dr. Albert E. Sloane, Dr. Claire L. Straith, Dr. A. Earl Walker, Dr. Henry Williams, and Mr. L. A. Watson.

This course is open only to qualified physicians and requests for registration should be addressed to: Dr. E. G. Gill, Gill Memorial Hospital, 711 South Jefferson Street, Roanoke, Virginia.

SOCIETIES

READING MEETING

The 84th meeting of the Reading Eye, Ear, Nose, and Throat Society was held October 27th. This was an "Academy Meeting." The following reported on courses taken in Chicago: Dr. John J. Penta, Dr. Harold L. Strause, Dr. Benjamin F. Souders, Dr. Philip R. Wiest, and Dr. Paul C. Craig.

AKRON ACADEMY MEETS

The Akron Academy of Ophthalmology and Otolaryngology had its first meeting on November 1st. The speaker was Dr. James H. Allen, professor of ophthalmology at the University of Iowa, who discussed, "Bacteriology of Infectious Diseases as They Involve the Eye, Internal and External." The paper was illustrated with lantern slides and was excellently done. A number of ophthalmologists from Cleveland and surrounding communities were present making a total attendance of 35. The following are the officers for the organization for the year 1948-1949: president, Dr. Vincent C. Malloy; vice-president, Dr. Clair E. Anderson; and secretary-treasurer, Dr. A. L. Peter.

WASHINGTON SOCIETY OFFICERS

New officers of the Washington, D.C., Ophthalmological Society are: president, Dr. Jerome A. Sansoucy; vice-president, Dr. J. Thomas Schnebly; secretary-treasurer, Dr. Thomas A. Egan; directors, Dr. Everett S. Caldemeyer and Dr. Edward J. Cummings.

On November 1st the society held a joint dinner meeting with the Baltimore Ophthalmological Society at the Kennedy-Warren Hotel in Washington. The next meeting will be held on January 3rd. The February meeting, date to be announced, will be held jointly with those attending the George Washington University postgraduate course. In March there will be another joint meeting with the Baltimore society, and the final program of the year will be presented on May 2nd.

PERSONALS

PROFESSOR JOSEPH MELLER

To celebrate Professor Meller's 75th birthday a collection of scientific articles is planned. Since at the present time contributions in German periodicals are necessarily limited in number, articles will be accepted for the *Festschrift* which have appeared in any periodical provided that the article was printed with the dedication to Dr. Meller's 75th birthday. Reprints of such original articles could then be bound in one volume and could serve as a *Festschrift*. Notice of such an article should be sent to Prof. J. Böck, Augenklinik, Graz, Austria.

Dr. Derrick Vail, professor of ophthalmology, Northwestern University Medical School, Chicago, gave The Snyder Ophthalmic Foundation lecture at the International Medical Assembly of the Interstate Post-graduate Medical Association of North America held at Cleveland, Ohio, November 9th to 12th. The subject of Dr. Vail's address was "Exophthalmos."

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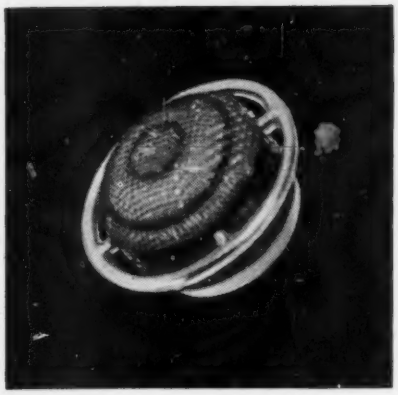

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